SERIES 3, Vol. 23, No. 2 FEBRUARY 1940

AMERICAN JOURNAL OF OPHTHALMOLOGY

CONTENTS

DEPARTMENTS

For complete table of contents see advertising page V

Subscription price in United States ten dollars yearly. In Canada and foreign countries twelve dollars. Published monthly by the George Banta Publishing Company, 450 Ahnaip Street, Menasha, Wisconsin, for the Ophthalmic Publishing Company. Subscription and Editorial Office: 640 S. Kingshighway, Saint Louis, Missouri. Entered as second class matter at the post office at Menasha, Wisconsin.

...the selection of a team-mate

Dear Bill:

Your disappointment couldn't have been greater than ours when you were unable to come home for the week end. Your mother had made one of her famous chocolate cakes for your special benefit. While the cake didn't exactly go to waste, we did wish you had been here to enjoy it with us.

Bill, I think I detect in your letter an indication of those misgivings that every young refractionist feels when he starts into practice. I remember I had the same apprehensions. But if you start right, there is nothing to fear in the future.

Right at this time you have a very important step to take in the selection of a team-mate, that is, a supply house whose reputation cannot be questioned. Your future professional reputation depends entirely upon your ability and the accuracy with which your prescriptions are filled.

Your refractive analysis is only as good as the material and workmanship that interpret it. That is why it is so important that you use good judgment in choosing your team-mate.

As you know, I chose Riggs many years ago, and I have never had occasion to regret it. They have been of valuable assistance to me in many ways, and I have learned I can always be sure that their standards of quality and precision are in accordance with mine.

Remember, Bill, there is no substitute for quality.

Affectionately,

Dad

RIGGS OPTICAL COMPANY

DISTRIBUTORS OF BAUSCH & LOMB PRODUCTS

AMERICAN JOURNAL OF OPHTHALMOLOGY
VOLUME 23 FEBRUARY, 1940 NUMBER 2

THE EFFECT OF THE INTRAVENOUS INJECTION OF NON-SPECIFIC PROTEINS UPON EXPERIMENTAL CORNEAL ULCERS*

EDWARD GALLARDO, M.D., AND RICHARD THOMPSON, M.D.
New York

The parenteral injection of foreign proteins is utilized extensively in ophthalmological practice. The reactions to such injections have been studied clinically and experimentally by a great number of workers. The large literature that has accumulated has been reviewed adequately[1, 2, 3, 4] so that only a brief summary is necessary here. It is generally recognized that the results of protein injections are a series of reactions involving practically all body tissues and fluids. These reactions are usually diphasic, the timing and magnitude of the phases depending upon the quantity of protein injected and upon the state of the recipient. The majority of the effects appear to result, directly or indirectly, from four principal reactions: fever, changes in vascular tone, changes in capillary permeability, and stimulation of leukocyte production (possibly a stimulation of the whole reticulo-endothelial system). The vascular changes have been ascribed, on doubtful grounds, to general sympathetic stimulation, followed by inhibition. The capillary permeability is first increased and then decreased. Probably secondary to these vascular changes are a number of other effects, including the early leukopenia, increased lymph flow, increased glandular secretion, and an increase in certain blood constituents (proteins, ferments, and specific antibodies).

When inflammatory lesions are already present, a marked increase in the degree of inflammation occurs following the introduction of the foreign protein. These so-called focal reactions appear to be accentuated local manifestations of the general reactions. Similar changes have also been described in supposedly normal tissues. Von Szily[5] produced capillary dilatation, exudation, and lymphocytic infiltration in the uveal tracts of normal rabbits by the parenteral injection of various proteins.

There has been much discussion as to which of the various reactions are responsible for the apparently beneficial effect of protein therapy upon certain infectious conditions. The majority of investigators have stressed the fever and leukocytosis as being most significant. Some organisms may be killed or attenuated by the temperature reached during the reaction,[6, 7] so that fever itself may be beneficial in some infections. A concomitant increase in the bactericidal power of the blood has been demonstrated.[8, 9, 10, 11] This is probably accounted for, in part at least, by the increase in leukocytes. A number of workers have found an increase in the opsonic power of the serum.[12,13,9] According to Bedson[13] this is not due to an increase in the normal opsonins but either to an increase in the alexin or to physico-chemi-

* From the Departments of Ophthalmology and Bacteriology, College of Physicians and Surgeons, Columbia University, and the Institute of Ophthalmology, Presbyterian Hospital.

cal changes favoring phagocytosis. Reimann[7] has shown that the enhanced fibrinogen content of the blood causes an increase in specific agglutination. The nonspecific reawakening of dormant antibodies may also play a part against some organisms. Whether the nonspecific β-lysins of the blood are increased has not yet been investigated.

It has also been suggested that beneficial effects of protein injections may result from nonspecific desensitization of sensitized tissues. There appears to be no experimental evidence supporting such a view although a nonspecific desensitization frequently follows protein injections (Isaacs[14]). Brown[15] has advanced the novel concept that protein injections produce their effect by a *nonspecific* "interfering" action of the *specific* antiprotein immune substances. This theory was based originally upon experiments with uveal allergic reactions produced by streptococci and was later extended to include intraocular and corneal infections. He produced marked inflammatory reactions of the uveal tract by the intravenous inoculation with streptococci of rabbits previously sensitized by intraocular injections of the same organism. These allergic reactions could be prevented or minimized in several ways. *Sensitization* was prevented if the sensitizing injections of streptococci were made into the eyes of typhoid-immune rabbits previously subjected to aspiration of the aqueous humor in order to increase the local agglutinin content. The *reactions* brought about by subsequent intravenous injection of the antigen could be cleared more rapidly than it could in controls, if they were produced in typhoid-immune rabbits possessing high agglutinin titers in the aqueous or if a parenteral injection of typhoid vaccine was given about the time of the reinjection of the antigen. A number of workers (Howard,[16] Allen,[17] and Key[18]) have reported beneficial clinical results in the treatment of corneal ulcers by protein administration. Experimentally Key[19] found some indication that such treatments would limit the size of staphylococcal ulcers in rabbits. Brown and Pugh[20] also found that staphylococcal corneal ulcers in rabbits could be favorably affected by nonspecific injections but, as mentioned above, referred the effect to the antityphoid agglutinins in the aqueous humor.

The work reported here consists chiefly of a comparison of the lesions produced by the intracorneal inoculation of staphylococci in normal rabbits and in rabbits which had received one or more intravenous injections of killed typhoid bacilli; either as "shocking" injections shortly before infection or as a series of "immunizing" treatments completed 10 days before infection. In addition, a number of observations bearing on the possible mechanism of the beneficial effect were made.

Methods

Albinos and mixed-breed rabbits of from 3,000 to 4,000 gm. were used in all experiments. Care was taken to distribute weights and breeds as evenly as possible through the experimental and control groups.

Typhoid injections: In all experiments but one the Goethals strain of B. typhosus was used. Eighteen-hour, agar-slant cultures were suspended in saline, standardized by turbidity measurements, and killed by heating to 60°C. for one hour. For immunization, one, two, or three series of intravenous injections were given up to 10 days before corneal infection, as noted in the protocols. The shock animals received one intravenous injection 18 to 20 hours before the corneal infections and, in one experiment, several times following infection. In ex-

periment 4 a vaccine* prepared so as to preserve the H antigen was used. The immunizing and shocking injections were arranged so that all the animals in one experiment were given the corneal infections at the same time.

Production and observation of corneal ulcers. For the production of corneal ulcers a typical, pathogenic, coagulase-producing staphylococcus isolated from a case of panophthalmitis was used. This is designated as staphylococcus A. Two experiments were done with the non-pathogenic white, coagulase-negative strain (called staphylococcus B) used by Brown and Pugh in their experiements. Eighteen-hour cultures on rabbits'-blood agar were suspended in saline and diluted to correspond with a barium-sulphate turbidity standard representing the required dosage. Injections of from 0.03 to 0.05 c.cm. were made into the central portion of the corneal stroma by means of tuberculin syringes and 27-gauge needles. Pontocain, 0.5 percent, was dropped into the conjunctival sac a short time previous to the injections. Experimental and control animals were injected alternately, and extreme care was taken to assure that all conditions were identical in the control and experimental groups. The number of staphylococci injected varied somewhat between experiments. The dosages of the A strain were between four and eight million organisms and of the B strain about 10 times these numbers. Since counts were judged by turbidity measurements, these figures are only approximate, but quantitative variations are not of great significance since control and experimental animals always were given as nearly as possible identical amounts of the same suspension.

Both eyes of the animals were utilized. The lesions were measured at frequent intervals over a period of several weeks. The later measurements are of scar tissue and not of active lesions. When the lesions were not circular the measurements given represent the average of two diameters. In addition the presence of general ocular inflammation was noted.

* Supplied by Eli Lilly Company.

Protocols of experiments

Corneal Infection with Staphylococcus A

Experiment 1. Five rabbits were immunized by two series of intravenous injections of heat-killed typhoid bacilli: 1st week, four doses of 150 million each; 2d week, four doses of 300 million each. Corneal inoculations were made 10 days after the last intravenous injections. Four control rabbits were included.

Experiment 2. Three rabbits were immunized by one series of five intravenous injections of heat-killed bacilli over a period of 10 days, receiving 2,100 million organisms in all. Three animals were "shocked" by one intravenous injection of 600 million heat-killed bacilli, 18 hours before corneal infection, and four similar injections during the eight days following. Corneal inoculations were made eight days after the last intravenous injection. Five control rabbits were included.

Experiment 3. Five rabbits were immunized by one series of five daily intravenous injections of heat-killed bacilli, receiving a total of 1,650 million organisms. Four rabbits were "shocked" by one intravenous injection of 300 million heat-killed bacilli, 18 hours before corneal infection. Corneal inoculations were made 10 days after the last intravenous injections. Four control rabbits were included.

Experiment 4. Three rabbits were immunized by three series of intravenous injections of H typhoid bacilli: in the 1st week, four doses of 100 million; in the 2d week, four doses of 200 million;

and in the 3d week four doses of 300 million organisms. Corneal inoculations were made 10 days after the last intravenous injections. Three control rabbits were included.

Corneal Infection with Staphylococcus B

Experiment 5. Three rabbits were immunized by three series of intravenous injections of heat-killed typhoid bacilli: 1st week, four doses of 150 million; 2d week, four doses of 300 million; and 3d week, four doses of 600 million. Three "shock" animals were given one intravenous injection of 450 million heat-killed typhoid bacilli 20 hours before corneal infection. Corneal inoculations were made 10 days after the last intravenous injections. Six control rabbits were included.

Experiment 6. Three rabbits were given one intravenous "shocking" injection of 450 million heat-killed typhoid bacilli 20 hours before corneal infection. Three control rabbits were included.

The results of these experiments are presented in graphic form. Each point on the graphs represents a single measurement in millimeters of the diameter of one lesion at the designated time following corneal infection. Measurements in the experimental and control groups are differentiated by the shape of the symbols as explained on the charts.

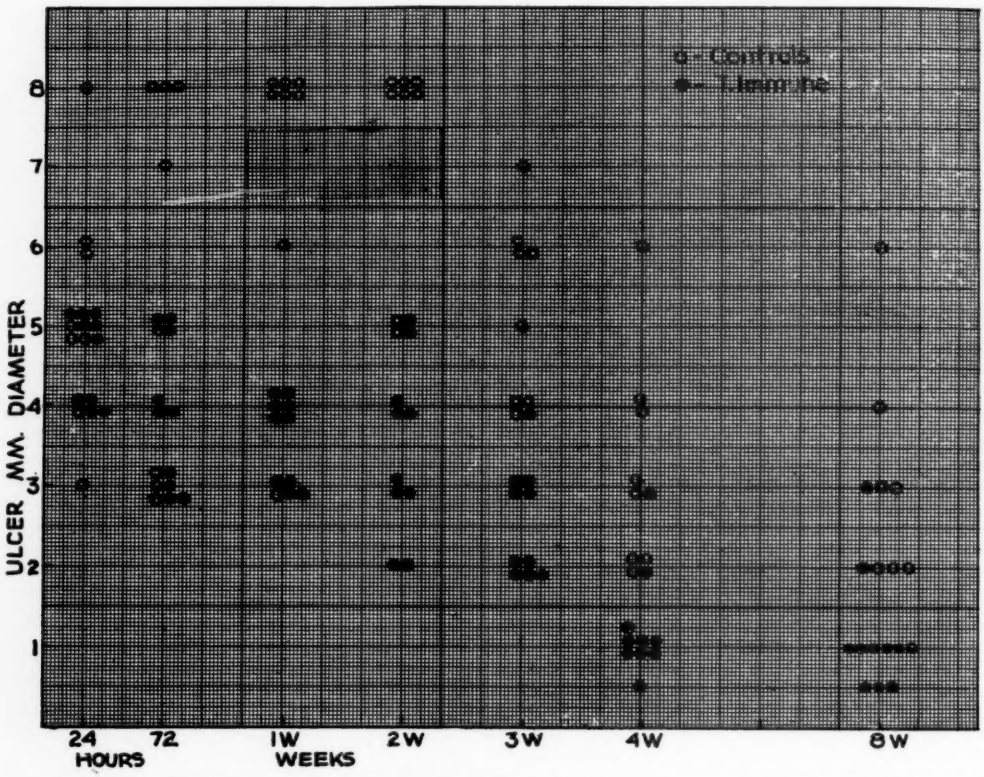


Chart 1 (Gallardo and Thompson). Experiment 1, measurements of corneal lesions in 5 immunized and 4 control rabbits. Corneal infection with Staphylococcus A, 10 days after last intravenous injection.

It will be seen that in all experiments but number 2 the corneal lesions of the

"immunized" and "shocked" animals were consistently smaller than those of corresponding controls. Not noted on the charts is the fact that the development of panophthalmitis was more frequent in the control groups: five control eyes developed generalized infection while only one in the treated groups did so.

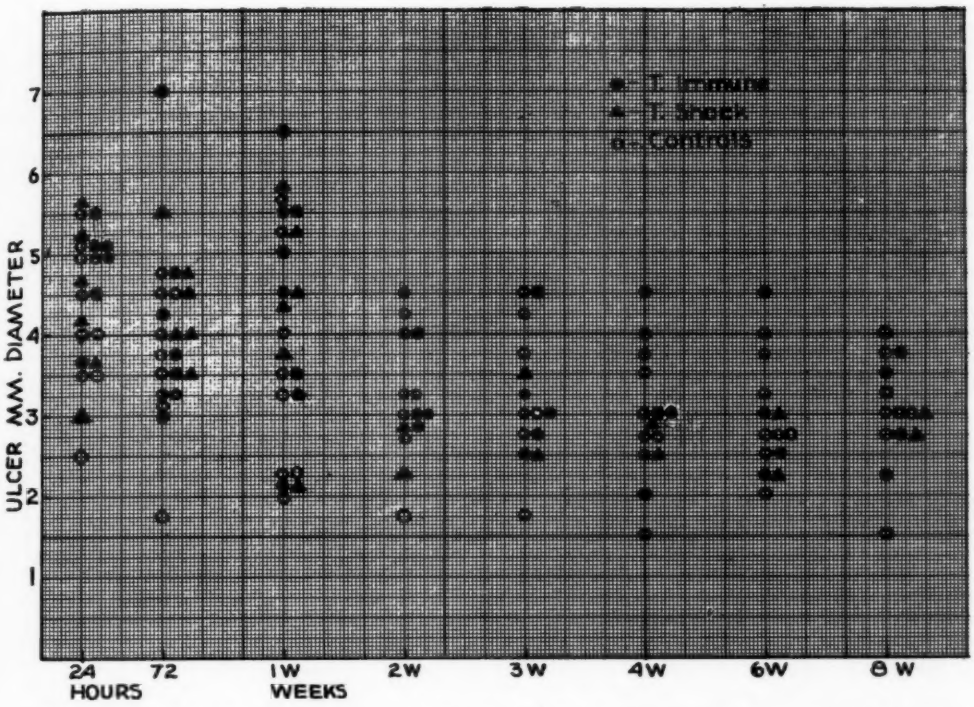


Chart 2 (Gallardo and Thompson). Experiment 2, measurements of corneal lesions in 3 immunized, 3 shock, and 5 control rabbits. Corneal infection with Staphylococcus A, 8 days after last intravenous injection.

Statistical analysis

Animals in individual experiments were not numerous enough to warrant a statistical expression of the differences in each experiment, but certain of the experiments were similar enough to warrant grouping for statistical analysis. The experiments with the A-strain staphylococcus were therefore grouped to compare the reactions in immunized and control animals. The shocked and control animals were likewise grouped in the B-strain experiments. The calculations were based on measurements made at the end of the second week after infection. The difference between the mean diameter of the lesions in the treated and control groups was determined and the standard error of this difference calculated from the formula:

$$SE = \sqrt{\frac{\sigma_t^2}{N_t} + \frac{\sigma_c^2}{N_c}}$$

Where $\sigma_t = \sqrt{\frac{\Sigma\delta_x^2}{N_t - 1}}$ and $\sigma_c = \sqrt{\frac{\Sigma\delta_x^2}{N_c - 1}}$

N_t = number of eyes in treated group
N_c = number of eyes in control group
δ_x = difference between individual diameter and mean diameter of group

Experiments 1 to 4 inclusive—Immunized and Control Animals. The mean diameter of the lesions in 30 control eyes was 4.42 mm.; the mean diameter of the lesions in 26 eyes in immunized animals was 2.85 mm. The difference is 1.57 mm. The standard error of difference is 0.424 mm. The difference is 3.7 times its standard error.

Experiments 5 and 6—"Shocked" and Control Animals. The mean diameter of lesions in 18 control animals was 2.4 mm.; the mean diameter of lesions in 12 shocked animals was 1.71 mm. The difference of means is 0.69 mm., standard error of difference is 0.3015. The difference is 2.2 times its standard error.

The statistical analysis supports the conclusion that both "shocked" and "immune" groups had some measure of protection against staphylococcal injections into the cornea. Judging from the charts, the shocking and immunizing injection had about equal effects. (The two groups were not directly compared in the statistical analysis.) The immunized animals carried high typhoid agglutinin titers (1:1000+) at the time of infection, while agglutinins could never be demonstrated in the "shocked" rabbits until 72 hours following intravenous injection of the vaccine (48 hours after corneal infection). The protection in the immune group is of interest since it has generally been assumed that the beneficial results of protein injections are limited to a short

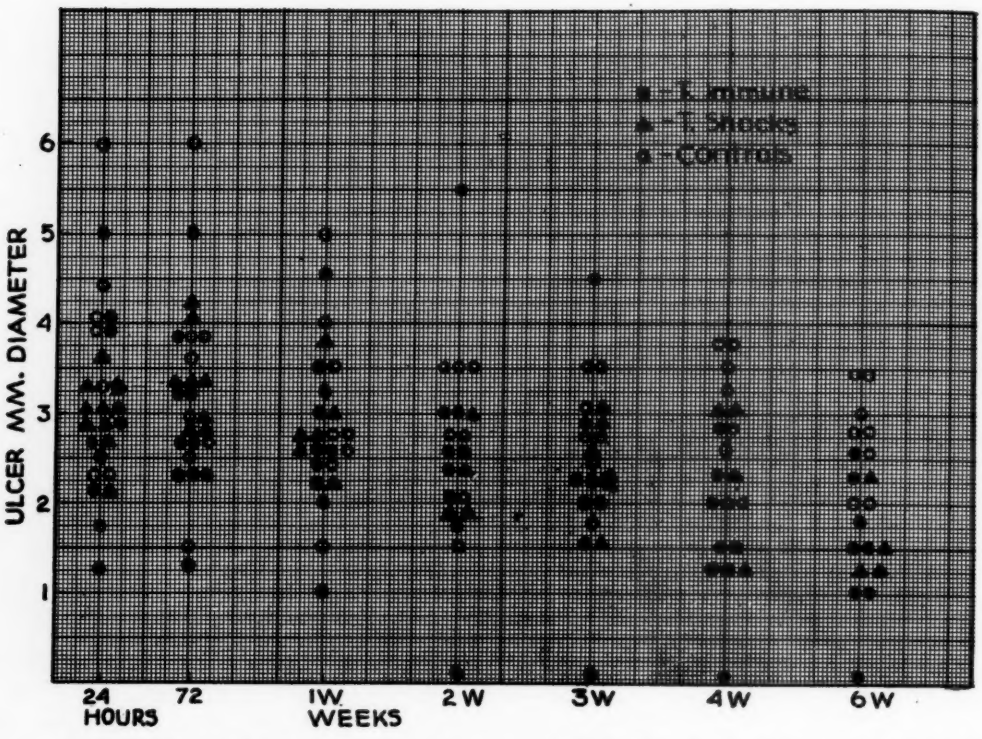


Chart 3 (Gallardo and Thompson). Experiment 3, measurements of corneal lesions of 5 immunized, 4 shock, and 4 control rabbits. Corneal infection with Staphylococcus A, 10 days after last intravenous injection.

period of time (24 to 48 hrs.). Several workers, however, have reported nonspecific protection lasting for periods of 10 days or more.[21 to 27] Phillippson[27] obtained maximum protection against dysentery organisms in mice by the reinjection of a nonspecific vaccine into mice previously immunized to the same vaccine. Protection was evident two hours after the injection and lasted in some cases as long as 22 days. As already mentioned, Brown found nonspecific protection against corneal infection persisting for 10 days and ascribed it to an interfering action of the antibodies.

Our chief purpose was to determine whether nonspecific protein injection would influence the course of corneal infections but a few observations and experiments were made which may have a bearing on the mechanism of such protection.

(1) As stated above the protection was apparently as great in the "shock" animals which had no demonstrable antibodies at the time of infection as in the immune animals which had high agglutinin titers.

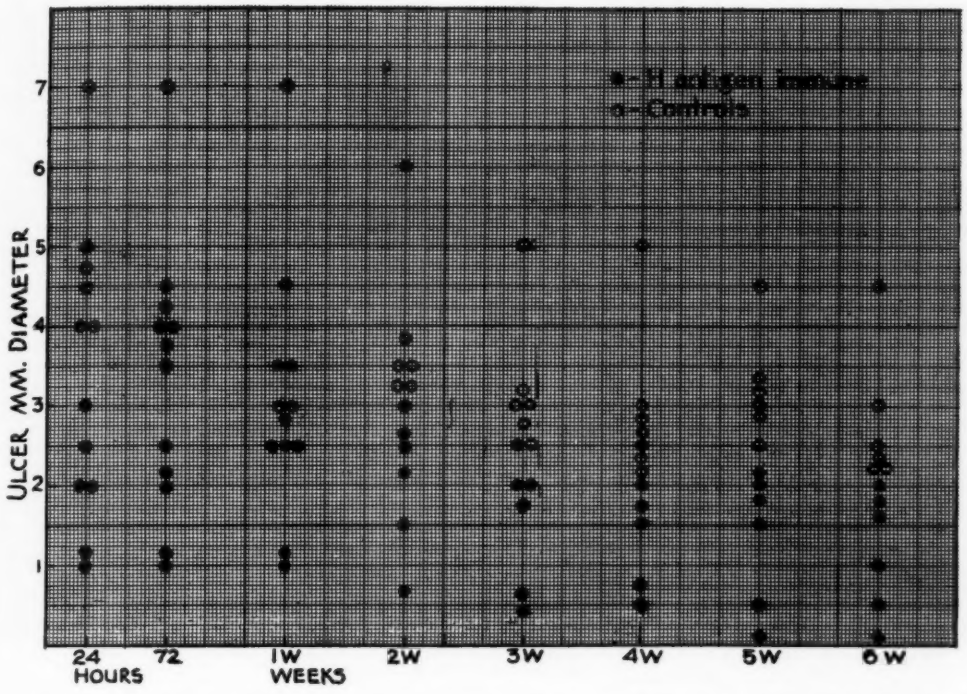


Chart 4 (Gallardo and Thompson). Experiment 4, measurements of corneal lesions in 3 immunized and 3 control rabbits. Corneal infection with Staphylococcus A, 10 days after last intravenous injection.

(2) Evidence was obtained that nonspecific protective factors other than specific antibodies were still increased 8 to 10 days after the immunizing injections.

White-cell counts. White-cell counts were made before and 8 to 10 days after protein injections but before the corneal inoculations were made. In all but three animals the counts increased and in many

cases were considerably higher at the latter period than before the injections. Of 18 animals, before any injections were given, the mean white-cell count was $6{,}533 \pm 492 \left(\sqrt{\frac{\Sigma\delta^2}{N(N-1)}} \right)$. The same 18 animals, 8 to 10 days after the last cells were made by separating two clean, sterile slides by narrow strips of vaselined paper so as to form three equal chambers having a depth corresponding to the thickness of the paper strips. One-tenth-cubic-centimeter quantities of three different broth dilutions of 18-hour cultures of the staphylococcus were

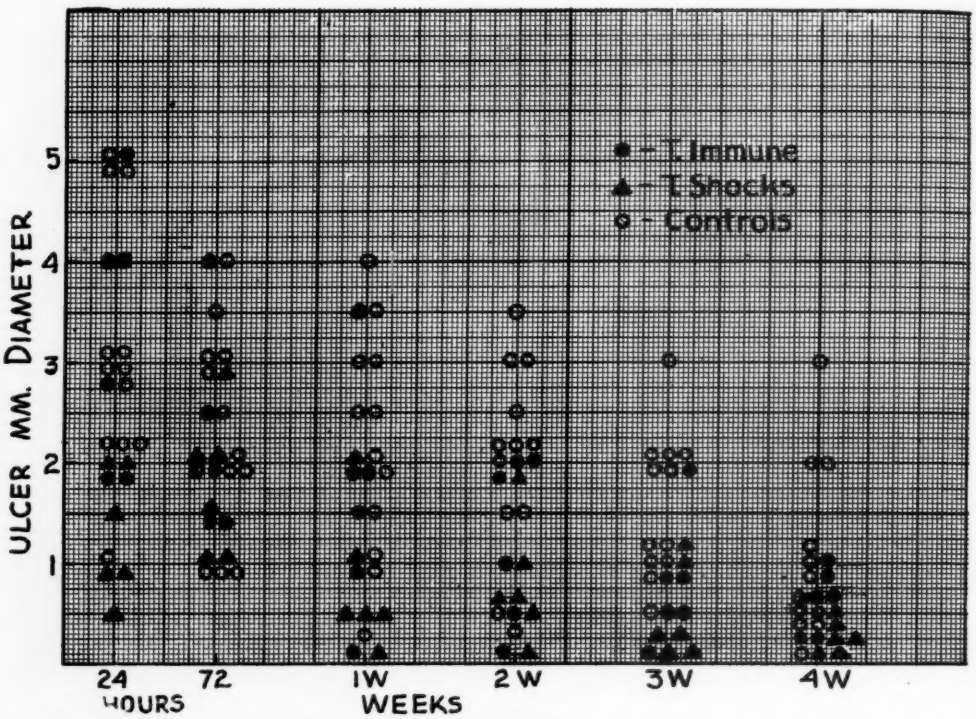


Chart 5 (Gallardo and Thompson). Experiment 5, measurements of corneal lesions in 3 immunized, 3 shock, and 6 control rabbits. Corneal infection with Staphylococcus B, 10 days after last injection.

of a series of immunizing injections, had a mean count of $9{,}080 \pm 500$. The difference between the means is 2,547 with a standard error of 730. The difference is more than three times its S.E.

Bacterial inhibiting power of blood. The growth-inhibiting power of defibrinated blood from normal, immunized, and shocked rabbits was tested against a pathogenic-type staphylococcus by the Wright slide-cell method.[11] The slide mixed with 0.9-c.c. quantities of the defibrinated blood. The dilutions usually found to be satisfactory were 1 to 200,000, 1 to 1 million, and 1 to 5 million. Measured amounts of the mixtures were allowed to run into the cells, which were sealed by brushing melted paraffin around the edges of the slides and incubated at 37°C. Three chambers were prepared from each dilution. The number of organisms inoculated into each cell was

TABLE 1

BACTERIAL INHIBITING POWER OF DEFIBRINATED BLOOD FROM NORMAL, SHOCKED, AND IMMUNIZED RABBITS. (EACH FIGURE REPRESENTS NUMBER OF ORGANISMS NECESSARY IN INOCULUM TO PRODUCE ONE COLONY IN CHAMBERS)

Normal	Shocked	Immunized*
2.3.3.5.5.7.7.8.9.10. 10.10.16.16.40.40.40.	65.460.500.600.600.2000.	50.50.50.130.150.180.
17 bloods tested	6 bloods tested	6 bloods tested

* Blood taken from immunized rabbits 8 to 10 days after the last intravenous injection and from shocked rabbits 18 to 24 hours following a single intravenous injection. In many cases the normal figures are of animals later shocked or immunized. In all experiments the shocked and immunized bloods were controlled by normal bloods tested at the same time.

determined by plating the broth dilutions. The staphylococci grew as definite colonies in the blood in the slide cells and could be readily counted with the naked eye. The results are shown in table 1. The numbers given are the numbers of inoculated organisms required to produce one colony in the chambers. They are the result of averaging the counts of the three chambers of each of the three dilutions.

It is evident that a marked increase in the inhibiting power of the blood against staphylococci occurred following a shocking injection of typhoid vaccine. This confirms the reports previously mentioned.[8, 9, 10, 11] The increases were not so marked in the immunized animals, some of the figures approaching the upper normals, but were still quite definite and show that the increase in inhibiting factors produced by protein injections is still present 10 days later.

To determine whether any part of the

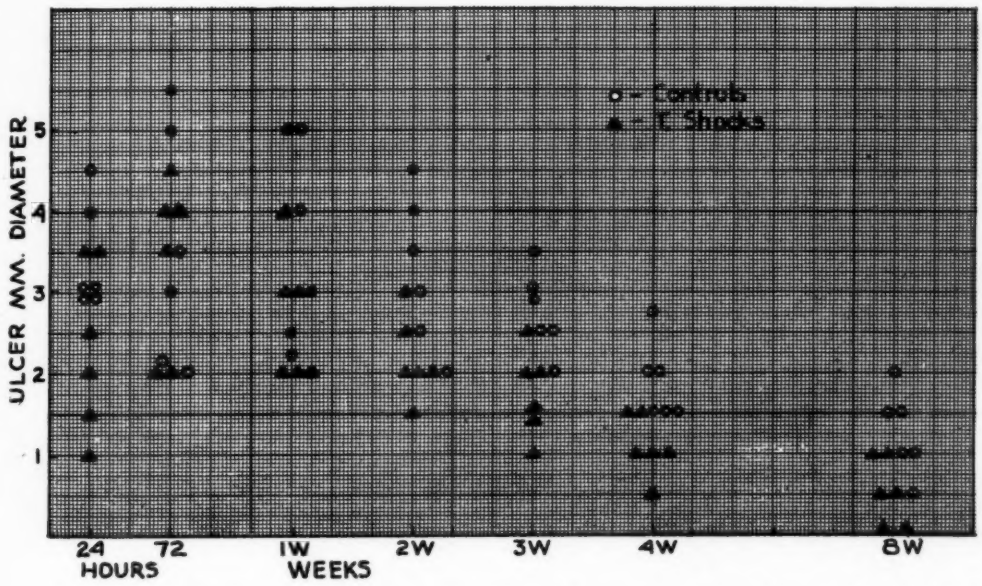


Chart 6 (Gallardo and Thompson). Experiment 6, measurements of corneal lesions of 3 shock and 3 control rabbits. Corneal infection with Staphylococcus B, 20 hours after single injection.

increase in inhibiting power was due to serum factors, the plasma was removed from defibrinated normal rabbits' blood and replaced by serum from "shocked" or "immunized" rabbits or by normal serum. The reconstituted bloods, centrifuged cells plus new serum, were inoculated with staphylococci as in the previous experiments and incubated in the slide cells. The colony counts in the "shock"- and immune-serum chambers were consistently about 50 percent of those in the normal-serum chambers, indicating that part of the inhibiting factors increased by the protein injections are contained in the serum.

Evidence was obtained in several experiments that the serum does not act entirely by its opsonic effect but exerts a direct inhibiting effect. The three types of serum—normal, shock, and immune—were incorporated (in 20-percent concentrations) into blood-agar plates. The plates were inoculated with equivalent numbers of staphylococci. Equal numbers of colonies appeared on all plates, but the colonies on the plates containing "shock" and immune serums were markedly smaller than those on the normal-serum plates (1.25 mm. diameter as opposed to 2 mm.) and were surrounded by definitely smaller zones of hemolysis (.25 mm. compared to 1.00). Inhibition of hemolysis in the "shock" or immune bloods has been noted frequently in the Wright-chamber experiments also. Other experiments still in progress and to be reported elsewhere indicate that the decreased hemolysis is not merely the result of the lessened growth of the organisms but results partly at least from a direct neutralization of the hemotoxin by some factor present in the "shock" and immune serums. The evidence available at present suggests that the so-called natural antitoxins are increased by the protein injections and that this increase also persists for a considerable time in the immunized animals.

Capillary permeability changes. A final observation should be mentioned. The first aqueous removed from the eyes of normal rabbits never clots. We found that the first aqueous removed from the eyes of "shocked" and immune rabbits formed very firm clots. Apparently the typhoid injections produced changes in capillary permeability which, in the case of the immunized rabbits, persisted for considerable periods (10 days) and which presumably could affect the course of ocular infection at that time.

Summary

Lesions produced by the intracorneal injection of staphylococci into rabbits were consistently and significantly smaller when the injections were made into the corneae of rabbits previously given intravenous injections of typhoid bacilli. The protection appeared to be equally great 20 hours following a single "shocking" injection of the nonspecific protein or 10 days following the immunizing injections when a slight but significant leukocytosis persisted. The nonspecific bacterial inhibiting power of the whole blood, the adjuvant power of the serum, and the direct bacterial inhibiting power of the serum all remained higher than normal in the immunized animals. The increase in capillary permeability resulting in the passage of fibrin into the aqueous fluid was also still present at the 10-day period.

It seems likely that all these factors play a part in limiting the lesions in the "shocked" and immunized animals and make it unnecessary to assume that the typhoid agglutinins *per se* are the protective factors.

635 West One Hundred Sixty-fifth Street.

References

[1] Peterson, W. F. Protein therapy and non-specific resistance. New York, Macmillan Co., 1922.
[2] Cecil, R. J. Jour. Amer. Med. Assoc., 1935, v. 105, p. 1846.
[3] Hektoen, L. Jour. Amer. Med. Assoc., 1935, v. 105, p. 1765.
[4] Woods, A. C. Allergy and immunity in ophthalmology. Baltimore, Johns Hopkins Press, 1933, p. 134.
[5] von Szily, A. Klin. M. f. Augenh., 1930, v. 85, p. 21.
[6] Carpenter, C. M., Oak, R., Mucci, L. H., and Warren, S. L. Jour. Lab. & Clin. Med., 1933, v. 18, p. 981.
[7] Reimann, H. A. Ann. Int. Med., 1932, v. 6, p. 362.
[8] Hahn, M. Arch. f. Hyg., 1897, v. 28, p. 312.
[9] Prausnitz, C., and Meissner, G. Zent. f. Bakt., I Orig., 1925, v. 94, p. 376.
[10] Wolff, L. K. Zeit. f. d. ges. Exper. Med., 1929, v. 67, p. 683.
[11] Wright, A. E., Colebrook, L., and Storer, E. J. Lancet, 1923, v. 1, p. 365.
[12] Ledingham, J. C. G., and Bulloch, W. Quoted by Bedson.[13]
[13] Bedson, S. P. Jour. Path. and Bact., 1915, v. 19, p. 191.
[14] Isaacs, M. L. Arch. Path., 1928, v. 5, p. 1028.
[15] Brown, A. L. Trans. Amer. Ophth. Soc., 1935, v. 33, p. 435.
[16] Howard, H. J. Amer. Jour. Ophth., 1928, v. 11, p. 685.
[17] Allen, T. D. Trans. Sect. Ophth. Amer. Med. Assoc., 1925, p. 135.
[18] Key, B. W. Amer. Jour. Ophth., 1927, v. 10, p. 666.
[19] ———. Amer. Jour. Ophth., 1926, v. 9, p. 351.
[20] Brown, A. L., and Pugh, J. Trans. Sect. Ophth. Amer. Med. Assoc., 1936, p. 123.
[21] Clark, P. F., Tellmer, C. E., and Stone, H. W. Jour. Inf. Dis., 1922, v. 31, p. 215.
[22] Nukada, S., and Kako, Y. Klin. Woch., 1929, v. 8, p. 2143; Zeit. f. Immunitätsfrsch., 1930, v. 67, p. 83.
[23] Nukada, S., and Anfuku, S. Ibid., 1931, v. 70, p. 1.
[24] Otsuki, S. Ibid., 1932, v. 74, p. 249.
[25] Nukada, S., and Fujii, K. Ibid., 1933, v. 79, p. 287.
[26] Nukada, S., and Okutani, M. Ibid., 1935, v. 86, p. 204.
[27] Phillippson, J. Acta Path. et Microbiol. Scandinav., 1937, v. 50, Suppl. 32.

SALZMANN'S NODULAR CORNEAL DYSTROPHY

Report of a case*

Everett B. Muir, M.D.
Salt Lake City, Utah

Duke-Elder[1] has defined this disease as "a rare, non-inflammatory and slowly growing progressive condition occurring in persons previously affected by phlyctenular keratitis, characterized by the development of nodules, showing at an early stage hypertrophic and at a later stage degenerative changes in the superficial corneal tissues."

It was first described by Salzmann[2] in 1925, when he published a report of 23 cases found in Austria. He thought the disease was more common than the scarcity of reports indicated.

Dewey Katz,[3] in 1930, described one case, and in 1935, with E. V. L. Brown,[4] two cases, with a review of the literature and pathological study. Their cases were the first to be reported in the United States.

Ladekarl[5] of Denmark, in 1930, reported two cases of "Atypical keratitis nodoso Groenouw" with an extensive review of the literature, including Salzmann's original paper.

Lugli,[6] in 1931, reported a series of nine cases in Italy.

Case report

W. G., a white male, aged 54 years, first came to the Eye Clinic of the Cincinnati General Hospital on July 18, 1938. He complained of failing vision.

Two years previously "white spots" had appeared on the right eye. These increased slowly in size and number, impairing vision. There had been a "film" over the left eye ever since childhood, rendering him unable to do more than distinguish lights with it. There was no history of inflammation, excessive lacrimation, or pain in either eye.

The examination showed a medium-sized man who appeared older than the given age. The upper-lid margins were thickened and slightly reddened, with a sparsity of cilia shared by the lower lids. There was slight injection of the lower tarsal conjunctiva of both eyes, and of the left bulbar conjunctiva.

Scattered over the cornea of the right eye were several grayish-white elevated nodes with semitransparent margins, very irregular in size and shape. They tended to form a rough circle around the pupillary area, and only one small node encroached on it. The slitlamp showed these nodes to be under the epithelium. They appeared to have involved Bowman's membrane and the superficial stroma. No vascularization was noted. The cornea between the nodes appeared clear.

The upper two thirds of the cornea of the left eye were completely occupied by a dense elevated leucoma whose irregular surface showed a shallow pit resembling a healed ulcer near the nasal limbus. Blood vessels ran into the leucoma from the whole upper two thirds of the limbus, but more especially from the nasal limbus. On the relatively clear lower third were a few small nodes, similar to those on the cornea of the right eye and unvascularized. The slitlamp showed the large upper node to have involved most of the corneal thickness, while the smaller nodes were superficial like those on the cornea of the right eye. Neither cornea stained with fluorescein.

In the right eye, the pupil, iris, and anterior chamber appeared normal. No

* From the Department of Ophthalmology of the Cincinnati General Hospital.

vitreous opacities were seen. A slightly hazy lens, combined with the irregular astigmatism produced by the nodes, rendered the fundus view indistinct. No gross fundus abnormalities were noted. The field of vision was within normal limits.

cornea of the left eye with a sharp plastic blade. There was little discomfort after the first postoperative night. Epithelization was complete three days later, medication consisting of atropine and 25 percent cod-liver-oil ointment.

While in the hospital he was found to

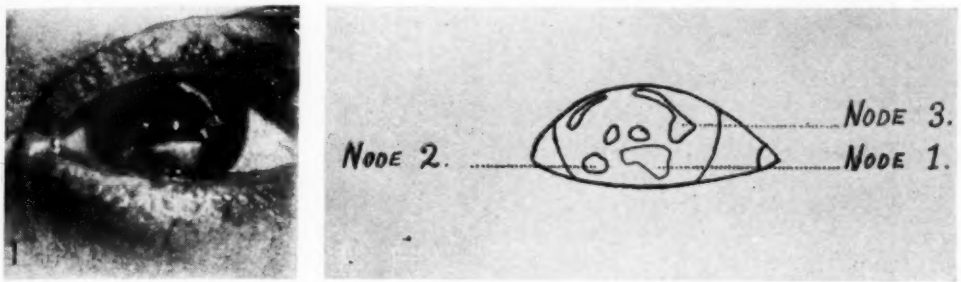


Fig. 1 (Muir). Right eye, showing the corneal nodes, and a diagram of the nodes. The principal nodes are numbered 1, 2, and 3 for descriptive purposes.

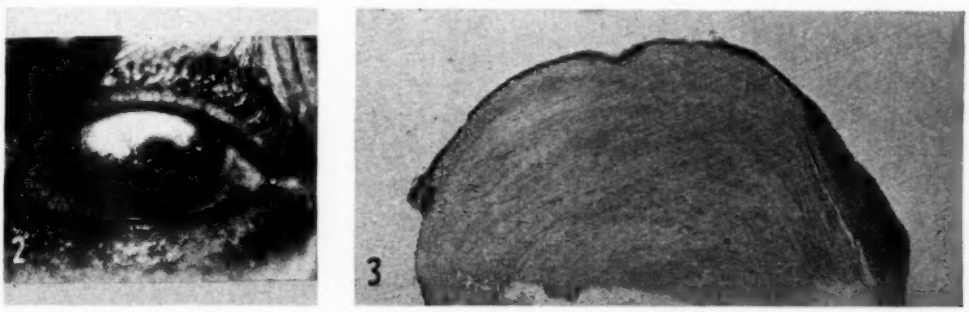


Fig. 2 (Muir). Left eye, showing the large leucoma on the upper portion of the cornea and the small ones below.

Fig. 3 (Muir). A low-power view of a section of node 1 from the cornea of the right eye, stained with hematoxylin and eosin. The thickened epithelium at one margin was lost during preparation.

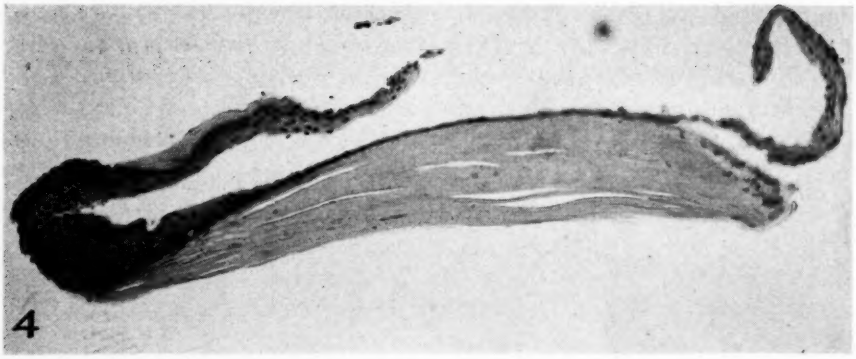
The anterior chamber and iris of the left eye showed no abnormalities of their visible portions. The pupil was concealed except for a very oblique-angled view from below. The condition of the lens, vitreous, and fundus could not be determined. Light projection and intraocular tension were normal. Vision, O.D., was 9/200, improved to 20/70 with a +9.00, D. sph. ⌯ + 2.50 D. cyl. ax. 120°.

Under cocaine anesthesia, the superficial layers of the large leucoma and all of the small nodes were peeled from the

have moderately severe diabetes mellitus of which he had been unaware.

The blood picture was normal, while the blood Kahn test was negative. A Mantoux test for tuberculosis with purified protein derivative was negative.

Six weeks later, when the operation on the left eye was deemed to have been justified by the lower third remaining clear, the nodes were peeled from the cornea of the right eye with the side of a keratome blade. Although there was a definite line of cleavage, an attempt was



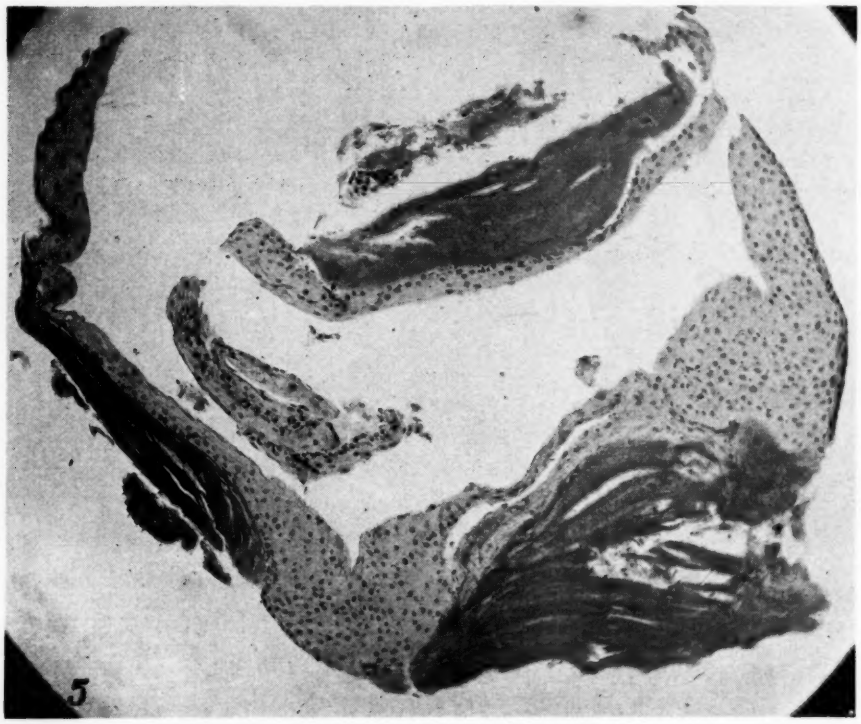


Fig. 4 (Muir). A low-power view of a section of node 2 from the cornea of the right eye, stained with hematoxylin and eosin.
Fig. 5 (Muir). A low-power view of a section of node 3 from the right cornea, stained with hematoxylin and eosin. This was really three nodes which had become confluent.

made to cut through normal underlying stroma. The postoperative reaction was greater than in the left eye. Ten days of treatment with hot boric-acid compresses, atropine, and 25 percent cod-liver-oil ointment were necessary to achieve healing.

Two weeks after operation, vision in the right eye was 20/200, improved to 20/70 with +9.00 D. sph. ⊂⊃ + 1.50 D. cyl. ax. 110°. At that time the slitlamp showed nebulous opacities at the sites of several of the previous nodes, and a few tiny nebulae scattered over the cornea. The left eye showed no sign of recurrence of the smaller nodes, while the large leucoma had not changed.

On April 5, 1939, the cornea of the right eye was clear, except for a few

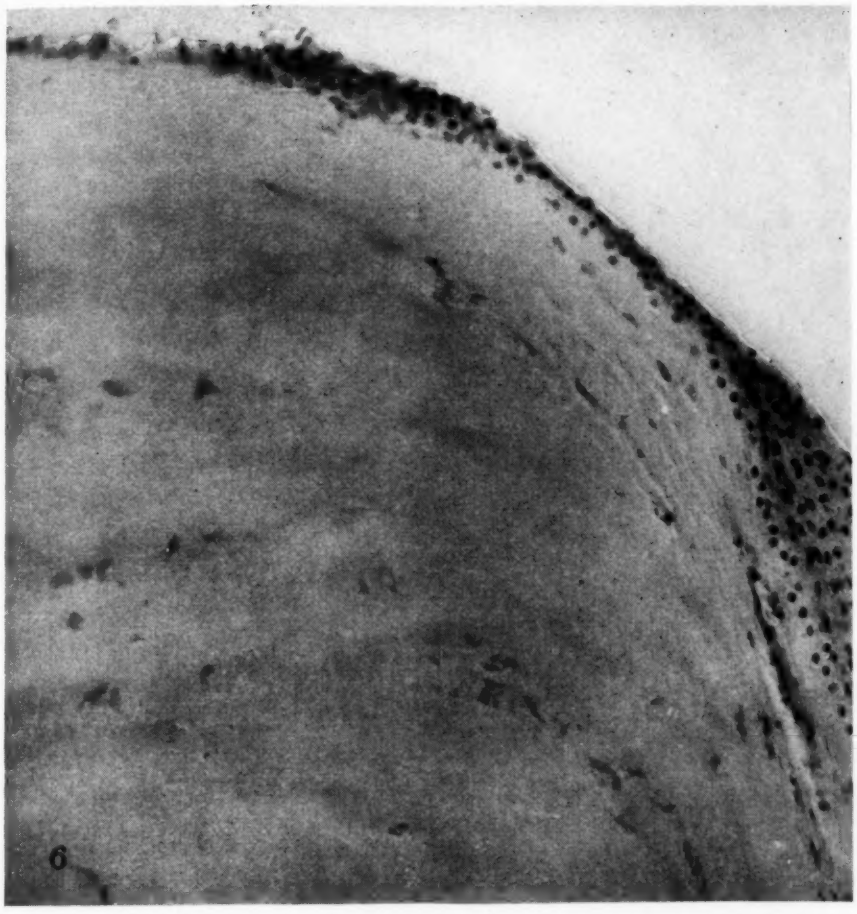


Fig. 6 (Muir). A high-power view of a portion of node 1, stained with hematoxylin and eosin, showing the island arrangement of the nuclei in the degenerate stroma.

small superficial nebulous opacities, chiefly at the sites of the former nodes. A better fundus view was afforded than at any previous examination. The lens and vitreous appeared clear. The fundus showed a mild grade of retinal arteriosclerosis, and two small, round, glistening, yellowish-white exudates near the macula suggestive of beginning diabetic retinitis. The large upper opacity on the cornea of the left eye was slightly smaller and slightly less dense, permitting a good red fundus reflex to be seen in the pupil on upward gaze. It continued to occupy the whole thickness of the stroma. A few small, superficial, very nebulous opacities were scattered over the lower portion.

Microscopic examination: Stained with hematoxylin-eosin and Van Gieson's stain, the low-power appearance of the nodes from the right eye was fairly uniform. Each had a rounded outline whose curvature depended upon its thickness.

The epithelium changed rather quickly from 10 to 25 irregular layers of cells at the margins, to 2 or 3 layers on the nodes. The cells in the thickest portion were irregular in size, shape, staining, and arrangement. The basal-cell layer was intermittent, and only occasionally recognizable as such. Many of the nuclei were pale and appeared partially disintegrated, while some were vacuolated. Scattered through and underneath the

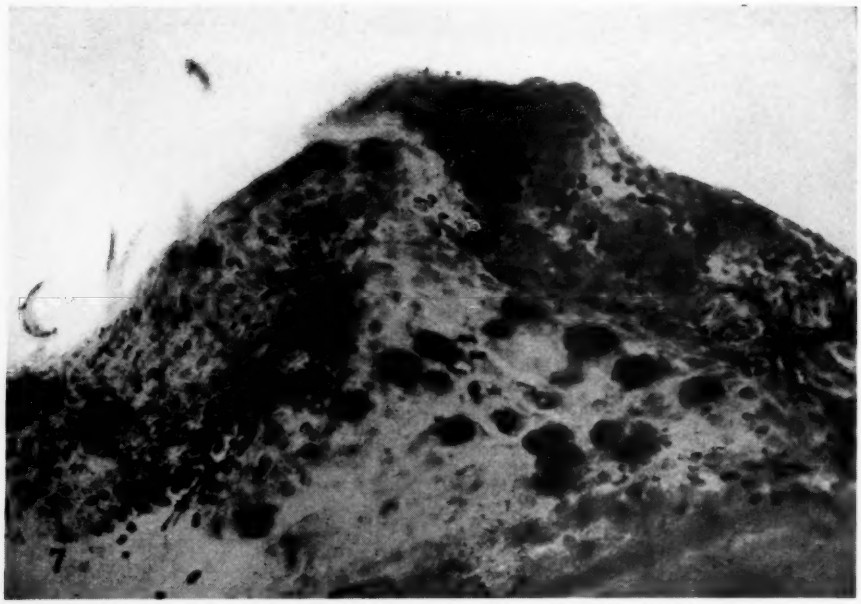
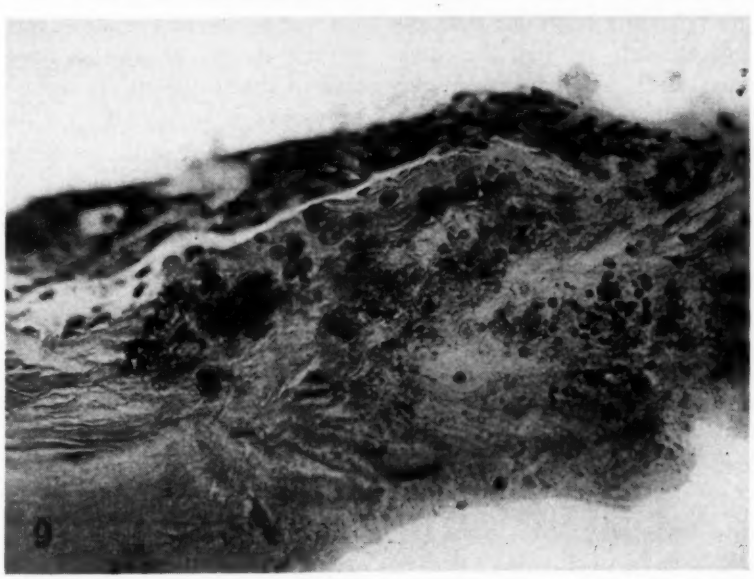


Fig. 7 (Muir). A high-power view of a section from the large node on the cornea of the left eye, stained by the "tri-chrome" method, showing some goblet cells.

thicker portions of the epithelium were a few lymphocytes and an occasional polymorphonuclear leukocyte. On the surface of the thicker epithelium of some nodes was a layer or two of long narrow cells with long, thin, pyknotic nuclei. The thinner epithelium usually consisted of either two or three layers of flattened cells with narrow spindle-shaped nuclei, or a layer or two of degenerate cells with indistinct boundaries, irregular in size, shape, and arrangement. In places no cell wall was discernible, with the cytoplasm a degenerate amorphous mass containing misshapen nuclei. On one node was an area where the epithelium was reduced to one layer of roughly polygonal cells with indistinct outlines and pale swollen nuclei. In one area of node number 2, the epithelium was represented by flat or spindle-shaped cells and nuclei intermingled with a remnant of Bowman's membrane.

Bowman's membrane appeared to have been destroyed over the nodes, except near the margins, where there were occasional short, wrinkled fragments. These fragments were often partly disintegrated, and fused imperceptibly with the epithelium and stroma. It was difficult to distinguish just where they began and ended.

At the margins of the nodes there was a gradual transition from normal corneal stroma to that of the nodes with its pale, swollen, usually scanty, nuclei, and poorly outlined swollen disintegrated fibers. The node fibers appeared to have been spread apart, and the interstices filled with a hyalinelike material which blended with the fiber structure so as to be indistinguishable from it in the deeper portions. In every node there was at least one area, and usually more than one, where the degenerative process was more advanced. In these areas, the fibers were either grossly distorted, or broken up into fragments, or both. The fragments were sometimes coarse and irregular, again finely granular, and yet again globular or roughly oval. In places, to a greater or less degree in all the nodes, there was



Figs. 8 and 9 (Muir). High-power views of sections from the large node on the cornea of the left eye, stained with hematoxylin and eosin. Figure 8 shows fine and medium-sized granules. Figure 9 shows granules varying from fine to coarse.

an invasion of clumps of lymphocytes mingled with fibroblasts and wandering cells.

Scattered through the stroma of each node were pale, swollen nuclei, evidently from the fibers. These were most numerous in node number 1. In this node also, these nuclei tended to be arranged in islands where an occasional lymphocyte or polymorphonuclear leukocyte could also be found. In some of these islands the nuclei showed a fine granular degeneration. This island arrangement of the nuclei was seen much less frequently in the other nodes.

Opposite the thickened epithelium at one margin of node number 1, between the distorted stroma fibers, was considerable amorphous material that resembled epithelial cytoplasm with Van Gieson's stain.

The stroma structure was best seen with Van Gieson's stain, while the epithelium and nuclei were best differentiated with hematoxylin-eosin.

The tissue from the large leucoma on the cornea of the left eye had the same general appearance as that from the nodes of the right except that it seemed to represent a more advanced stage of the pathological process. An occasional whorllike arrangement of the epithelium suggested a tendency to cornification, while in some of the thicker portions was a fine, granular degeneration. In several places a few goblet cells were noted.

No remains of Bowman's membrane were seen.

The stroma showed extensive collections of lymphocytes in which were mingled a few polymorphonuclear leukocytes and fibroblasts, suggesting a chronic inflammation. The numbers of corneal nuclei varied inversely with the degree of degeneration.

The granular areas were larger and more plentiful than those in the nodes on the cornea of the right eye. The granules varied from fine to coarse in some areas. Other areas contained only very fine granules, and yet others contained many large oval, round, or irregular bodies, staining more deeply than the fiber remnants and hyalinelike material. The smaller nodes were apparently of more recent formation, and similar to those of the right eye.

Summary

A case of bilateral Salzmann's nodular corneal dystrophy is presented. The occurrence of the large node on the left eye antedated the smaller nodes on the right by nearly 50 years. The appearance of the small nodes on the left, both before removal and microscopically, suggested their age to be approximately that of the nodes on the right eye. While previous phlyctenular keratitis might have played a role in the etiology of the large vascularized node on the cornea of the left eye, there was no evidence of this factor to be found on the right.

The treatment advocated by previous essayists, surgical removal of the nodes, was used and found effective.

The writer is indebted to Dr. Mary Knight Asbury for helpful advice in the preparation of this report.

1120 Boston Building.

References

[1] Duke-Elder. Text-book of ophthalmology, v. 2, p. 2019.
[2] Salzmann, M. Zeit. f. Augenh., 1925, v. 75, p. 92.
[3] Katz, D. Arch. of Ophth., 1930, v. 4, July, p. 16.
[4] Brown, E. V. L., and Katz, D. Arch. of Ophth., 1935, v. 13, April, p. 598.
[5] Ladekarl, P. M. Acta Ophth., 1930, v. 8, p. 213.
[6] Lugli, L. Boll. d'Ocul., 1931, v. 10, p. 216.

SYPHILITIC PRIMARY OPTIC ATROPHY

II. General considerations and the results of treatment by standard methods (especially subdural treatment and induced fever). A critical review*

Joseph Earle Moore, M.D. and Alan C. Woods, M.D.
Baltimore, Maryland

Introduction

In 1932[1] one of us (J.E.M.) brought up to that date the literature on syphilitic primary optic atrophy. Recently (1940) we[2] have presented a critical review of the pathology and pathogenesis of this condition. In this paper we present a further review, dealing with more general considerations, especially incidence, diagnosis, and the results of conventional forms of treatment (tryparsamide, subdural treatment, malaria, electropyrexia, or mechanical fever) of syphilitic primary optic atrophy. These several reviews are preliminary to the publication of a detailed study of our own clinical material.

General considerations

Since the publication of the 1932 review,[1] several papers of more or less general interest have appeared. Zeeman,[3] on the basis of three cases has discussed the occasional extreme difficulty of differential diagnosis between tabetic optic atrophy and certain other conditions. Comauer[4] published a lengthy article which summarizes some of the older literature and provides a classification of the optic atrophies in general, but adds nothing new. Still more recently, Schwarzenburg[5] presented a similar review.

Marinesco, Draganesco, and Grigoresco[6] have reported an instance of the relatively rare association of optic atrophy with Erb's syphilitic spinal spastic paraplegia, in which necropsy showed an adhesive chiasmal arachnoiditis, Monterro Sales[7] reported a statistical review of 23 cases of optic atrophy, which adds no new information. Jefferson[8] emphasized the fact that optic atrophy may be due to compression of the chiasm and optic nerves by intracranial aneurysms. His long paper reports 12 personally observed cases of intracranial aneurysms affecting the visual pathways and analyzes 66 previously reported cases. There is no mention of syphilis as a cause of intracranial aneurysm, and, as a matter of fact, it is probably an uncommon cause.

The frequency of syphilis as a cause of primary optic atrophy

Moore[9] brings up to date the etiologic study of optic neuropathies from the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital. His series, combined with those previously reported from the same source, now includes 373 patients, of whom 129 had primary optic atrophy. Syphilis was the etiologic factor in 51 percent of these, brain tumor in 18 percent, the remainder scattered among arteriosclerosis, disseminated sclerosis, toxic amblyopia, and undetermined causes.

The visual fields in syphilitic primary optic atrophy

Sloan and Woods[10] have presented an important and exhaustive study of the visual fields of 103 syphilitic patients who showed evidences of involvement of the optic pathways. Of these, 56 patients had primary optic atrophy; 12 showed normal fundi but nevertheless defects in

* From the Syphilis Division of the Medical Clinic and the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

the visual fields similar to those found in frank primary atrophy; 12 showed homonymous field defects with a fundus picture either normal, showing questionable pallor, or retinal arteriosclerosis; and 23 had either active or preëxisting optic neuritis. Very careful perimetry was performed by a single observer. The authors reviewed the older literature and pointed out that in syphilitic primary optic atrophy, central scotomas are stated to be exceedingly rare (about 2 percent). Their own studies, however, showed that in primary optic atrophy, the field defects were of four separate types: (1) concentric contraction of the peripheral field associated with late loss of visual acuity (12 percent); (2) sector-shaped, or nerve-bundle defects, with which loss of vision might be early or late, depending on involvement of the papillomacular bundle (34 percent); (3) central or cecocentral scotoma with normal peripheral fields, associated with early loss of visual acuity (14 percent); and (4) central or cecocentral scotoma with defects in the peripheral fields, also associated with early loss of vision (39 percent). Contrary to the statements of earlier workers, a central or cecocentral scotoma was found in 53 percent of cases with syphilitic primary optic atrophy.

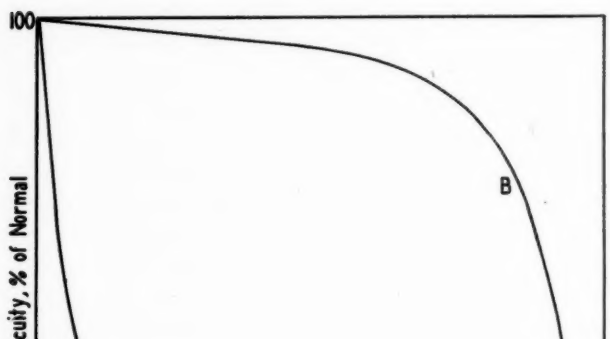


Fig. 1 (Moore and Woods). Hypothetical curves showing rate of loss of central visual acuity in (A) patients with central or cecocentral scotoma and (B) patients with peripheral constriction of the visual fields only.

This finding is of great importance for an understanding of the apparent rate of failure of visual acuity in optic atrophy, and may be expressed diagrammatically as in figure 1. In hypothetical patient A, with a central or cecocentral scotoma, the initial rate of failure of central visual acuity (that is, useful vision) may be very rapid, with subsequent much slower progression as the peripheral fields gradually constrict. In patient B, on the contrary, with peripheral contraction only, the atrophic process may progress for some time before the patient is aware of any visual impairment. In the one case, the diagnosis of optic atrophy may be made relatively early; in the other, relatively late. These two major types of failure of visual acuity must be given due weight in considering the effects of any form of treatment.

Sloan and Woods make a number of other important observations, as follows: In general, in any patient the fields of the two eyes show a marked similarity, though the defects are frequently more advanced in one eye than in the other. Defects for red practically always precede those for blue.

Changes in the fields are probably the

earliest clinical evidence of syphilitic optic atrophy, since field defects were present in all patients who showed any ophthalmoscopic pallor of the nerves or any reduction in the corrected visual acuity; and since in three patients who originally showed field defects only, definite optic atrophy later developed. The diagnosis once made, studies of the fields provide a more reliable method of following the course of optic atrophy than does either ophthalmoscopic examination or determination of visual acuity.

From the visual-field findings, Sloan and Woods believe that in primary optic atrophy the lesion probably lies in the optic nerve itself rather than in the chiasm or posterior to it, and is probably a peripheral and interstitial neuritis associated with secondary degeneration of the nerve fibers.

As to the question whether apparent chiasmal defects in the fields suggest the possible diagnosis of optochiasmal arachnoiditis,[2] there were included in the material of Sloan and Woods no outspoken examples of binasal or bitemporal hemianopsia, although in 13 of 50 patients the findings were such as to suggest that these types of field defects might previously have been present or were on the point of developing. It is of particular interest, however, that in 10 of 12 patients with homonymous field defects there were associated neurologic lesions, probably vascular, localizing the damage well posterior to the chiasm, and therefore almost surely not due to chiasmal arachnoiditis.

Dark adaptation in the early diagnosis of primary optic atrophy

The light minimum, or smallest amount of light that can be distinguished may be measured: (1) in an eye adapted to a specified level of illumination (photopic vision, probably dependent largely on cone function); (2) in the completely dark-adapted eye (scotopic vision, probably dependent chiefly on rod function); or (3) in an eye that has been in the dark for a shorter period of time than that required for complete dark adaptation. A determination of the so-called curve of dark adaptation involves all three of these types of measurements. The curve shows graphically the rate at which the light minimum decreases (that is, the sensitivity to light increases) as the initially light-adapted eye becomes dark adapted. In addition to the state of adaptation of the eye, two other factors have a marked influence on the light minimum: (1) the size of the pupil, and (2) the region of the retina tested. Inadequate control of one or more of these three variables is probably responsible for the conflicting results obtained by the various investigators who have used this form of test in studying patients with neurosyphilis.

Among 107 tabetics, Behr[11] (1915) found three with an isolated defect in the light minimum of the dark-adapted eye, associated with normal fundi, vision, and visual fields. These patients in later years developed pallor of the discs, loss of central visual acuity, and defects in the visual fields. He recognized the possible influence of miotic pupils in the test, and in such cases used artificial means to dilate the pupils before making the light-sense measurements. It is questionable, however, whether by the use of a mydriatic the Argyll Robertson pupil can be dilated to the size of the normal pupil in complete darkness.

Igersheimer[12] (1919) pointed out that Behr's three patients did not show unequivocal evidence of an isolated defect in the light sense associated with normal fundi, fields, vision, and pupillary reactions. From his own studies of neurosyphilis, Igersheimer concluded that the

light minimum in scotopic vision may remain normal in spite of marked reduction in vision and fields, and that therefore such tests are not of great value in differential diagnosis.

Schindler[13] (1922) found a marked decrease in the light sensitivity of the dark-adapted eye in all patients with manifest tabetic atrophy and in one patient with normal optic nerves. She found, however, that the light minimum varies with the size of the pupil and with the attention and intelligence of the patient, and concluded that such tests are therefore of little practical value in the early diagnosis of tabetic optic atrophy.

Gasteiger[14] (1927) found that in the dark-adapted eye, the light minimum was invariably affected in all patients with definitely established tabetic optic atrophy. In all cases examined, the pupils were dilated either with atropine or homatropine.

Accardi[15] (1933) used the Negel adaptometer to test the scotopic light minimum in a paracentral region of the retina. He examined 14 patients who had tabes, taboparalysis, or dementia paralytica, and found reductions in light sensitivity which he believed were too great to be caused by miotic pupils. He concluded, therefore, that such tests may be useful in the early diagnosis of neurosyphilitic affections.

Rutgers[16] (1923), although he did not test patients with tabetic primary optic atrophy, has contributed data on the effect of size of pupil in light-sense determinations. His results showed that when the pupil is either dilated with atropine or contracted with pilocarpine, the curve of dark adaptation is influenced in two different ways. The size of the pupil has not only a direct effect on the magnitude of the light minimum, but also an indirect effect in that it influences the level of previous light adaptation.

The possibility of regional differences in light sensitivity has received little attention in those investigations in which neurosyphilitic patents were studied. The importance of this factor has, however, been pointed out in a number of other investigations. Leibrecht[17] (1898) mentioned the possibility of a normal-adaptation curve in the central retina with an abnormal adaptation in the periphery, or vice versa. Lohmann[18] (1906) pointed out that tests of the scotopic light minimum, made without control of fixation, measure only the sensitivity of the parafoveal retina and therefore would not detect abnormalities either in the macular region or in the far periphery. Stargardt[19] (1909) showed that in pathologic conditions the scotopic light sensitivity of the eye can be different in different parts of the retina. Wölfflin[20] (1920) also emphasized that in localized pathologic processes, a true picture of the defects in sensitivity to light can be obtained only by making tests in a number of different regions of the retina. However, Rutgers[21] showed that when the curve of dark adaptation is determined without control of fixation, the results depend upon the regional location of the pathologic process as determined by visual-field studies.

These various studies of light sense in tabetic optic atrophy suggest a number of problems that require investigation. Further studies are needed in order to determine whether in early tabetic optic atrophy there is a generalized decrease in light sensitivity throughout the retina, or whether such a defect may be expected only in those regions in which perimetric studies also show functional loss. Studies are also needed of both photopic and scotopic light sense and of the rate of dark adaptation in order to determine not only if these tests are of value, but which is of the greatest value in the early diagnosis of tabetic optic atrophy. In such studies, account should be taken of all

the variable factors that may influence the results. In particular, the effect of pupillary size must be taken into consideration because of the frequency of miotic pupils in tabetics.

Moreover, the demonstration, since the publication of all these papers, that failure in dark adaptation in nonsyphilitics may be definitely associated with vitamin-A deficiency, indicates that this factor also should be considered during the next investigations in this field.

The early diagnosis of primary optic atrophy

It is possible that further studies of the light sense in neurosyphilitics may give some indication of impending degeneration in the optic nerves. However, until some such further test is discovered, it is clear that the early diagnosis of primary optic atrophy will rest on a procedure advocated by one of us (J.E.M.) in 1932;[1] that is, the routine ophthalmologic examination of every patient with neurosyphilis, and especially of those with tabes. This should be done whether or not the patient has visual complaints; and since, as Sloan and Woods point out,[10] the earliest sign of optic atrophy is to be found in field defects, it must include careful perimetry.

However, Foroni[22] suggests that early diagnosis may be made by another method, which he bases on the observation of Cohnheim that a normal blood vessel reacts to a compression stimulus by an increase in its width as soon as the pressure is removed, with a return to normal after a short period. An inflamed blood vessel, however, requires much more time to return to its original width. The eyeground is constantly observed during a period in which pressure (pressing the eyeball from behind forward) is exerted upon the eyeball and then released. While pressure is being exerted, the central artery should continue to pulsate. When, after several moments, the pressure is suddenly released, it will be noted that in the presence of a latent inflammatory process the papilla, which was very pale during the compression, appears "filled and swollen," due to the appearance of many at-first-invisible capillaries. There is a noticeable widening of the central vessels, particularly of the veins. This is the picture of a definite optic neuritis. Gradually, after several seconds to one minute, there is a return to normal. In the absence of neuritis the vessels show only a simple, transitory congestion or hyperemia of the papilla that lasts for 10 to 20 seconds. In the case of optic atrophy the reaction is very slight and transitory, never lasting more than 10 seconds. The value of such a test is, however, questionable. It depends to such a large extent on fine gradations of timing and the personal equation of the observer that the results are open to obvious criticism.

The effect of trivalent arsenical drugs on the optic nerves

As pointed out in Moore's (1932) paper,[1] earlier workers with optic atrophy were opposed to antisyphilitic treatment in any form. It was believed that such treatment, particularly with the arsenicals, increased the rate of progress of the degenerative process in the optic nerve. This blanket incrimination of all arsenicals grew out of the toxic effects of atoxyl on the optic nerve in trypanosomiasis. The antiarsenic dictum was still present in 1933, when La Croix[23] expressed the opinion that tryparsamide should not be used except in desperate cases of neurosyphilis, and that any arsenical compound is contraindicated in tabes and optic atrophy. He believed that

the optic nerve is selectively susceptible to the toxic action of all arsenical drugs, that arsenic makes optic atrophy worse, and that it may cause a central scotoma. Modern textbooks on ophthalmology (Berens),[24] on the other hand, recommend the arsenical treatment of optic atrophy.

Even yet, however, there still persists among an older group of ophthalmologists the superstition that the trivalent arsphenamines may damage the optic nerves. That this is superstition unfounded in fact has been amply demonstrated by the administration of uncounted millions of injections to many millions of patients in the past 30 years. The complete innocuousness of the arsphenamines for the optic nerves is not in the least shaken by the curious report of Skirball and Thurman.[25] These workers claim to have observed the development of optic neuritis (sometimes followed by secondary atrophy) after the administration of an arsphenamine in 20 patients (19 of whom had early syphilis); and this phenomenon occurred in the astoundingly high proportion of 2.7 percent of all patients with early syphilis treated. The neuritis subsided when arsphenamine was discontinued. This report, at direct variance with the experience of syphilologists and ophthalmologists the world over, has never been confirmed; and it is entirely probable that the authors were dealing with an optic neuritis produced by too little, not too much, arsphenamine (that is, a syphilitic ocular or neurorecurrence).

The results of conventional forms of treatment

As we have recently pointed out,[2] the course of untreated syphilitic primary optic atrophy is to permanent and complete blindness within 3 years in 70 percent of of all cases, within 5 years in 90 percent, and within 7 to 9 years in all except the very rare case. It is obvious, from these data, that no system of treatment can be regarded as successful unless the observation periods after treatment are long enough to justify conclusions and unless the development of blindness can be prevented or slowed beyond the limits mentioned in a significant proportion of patients.

In Moore's 1932 review,[1] it was concluded that: (1) tryparsamide was absolutely contraindicated in the treatment of the syphilitic optic atrophies; (2) subdural treatment had been found to be of some value in arresting the atrophic process in about 50 percent of patients with early atrophy; (3) fever therapy, chiefly with malaria, had also been found to be of some value, but, in view of conflicting reports, was to be regarded as still in the experimental stage.

The past seven years have offered additional evidence on these three latter points, which we now examine.

Tryparsamide in the treatment of optic atrophy

The pentavalent arsenical drug tryparsamide has been amply demonstrated by many observers (most recently and thoroughly by Sloan and Woods[26]) to cause toxic amblyopia with resultant partial or complete atrophy, depending on the extent to which the drug is pushed following the appearance of objective visual-field evidence of nerve damage. The incidence of this objective reaction in 2,087 tryparsamide-treated cases gathered by Sloan and Woods (excluding all those known to have preëxisting optic atrophy) was 3.53 percent. The field defects are quite characteristic, with vertical and nasal constriction and relative sparing of the temporal fields, without scotomas, and can usually be differentiated from the field defects of syphilitic optic atrophy.

Sloan and Woods draw no conclusions as to the advisability of use of the drug in patients with preëxisting optic atrophy; but from their paper and from the review of Wagener[27] may be gathered the incidence of objective tryparsamide reactions in 104 cases from the literature in which the drug was received in the presence of preëxisting ocular damage (usually optic atrophy, though this is not always clearly stated). Thirty-six of these 104 patients developed objective visual reactions, an incidence of 34.6 percent (that is, ten-fold the incidence in patients with previously normal eyes). Included in this group are the 27 patients with optic atrophy purposely treated with tryparsamide by Cady and Alvis[28] and the 21 with optic atrophy treated by Lees.[29] The report of Lees is of special interest, since he concluded that the tryparsamide treatment of optic atrophy is beneficial. Of his 21 patients:

> in 4, the drug was discontinued because of severe subjective symptoms;
> in 2, there was rapid deterioration of vision, apparently due to direct toxic action of the drug;
> in 3, the progress of the atrophy was apparently not affected;
> but in 12, the atrophy was apparently arrested for periods ranging from 6 months to 5½ years.

Not included among the 104 patients mentioned above are the 10 cases with reputed optic atrophy treated with tryparsamide by Mayer.[30] On the basis of the data supplied by him, the diagnosis of syphilitic primary optic atrophy cannot be accepted in 4 of his 10 cases [in cases 1, 7, and 8, the diagnosis was apparently made on the untenable basis of color of the discs alone, since visual acuity and fields were essentially normal; in case 3, in which visual acuity before treatment was said to be 20/200 and 20/100, respectively, the fields are described as normal (an association which appears to be impossible, if the visual failure was due entirely to optic atrophy), and after treatment, visual acuity rose to 20/20 in each eye, a degree of improvement never before reported in primary optic atrophy by any observer with any method of treatment]. Moreover, in two further cases (4 and 9), the optic atrophy was apparently unilateral, an unusually favorable type for treatment. Nevertheless, there was no objective damage from tryparsamide in any of his 10 patients, including the 4 in whom the diagnosis of bilateral primary optic atrophy seems acceptable. Furthermore, there was apparent arrest or even improvement in all, over periods of time ranging from 3 to 9 years. Both Mayer himself, and Lillie in the discussion of his paper, draw the conclusion that "tryparsamide is not more dangerous from the visual standpoint than other preparations used in the treatment of neurosyphilis in general or optic atrophy in particular."

It seems clear, on the basis of the experience of Cady and Alvis, Lees, and Mayer, that Moore's 1932 absolute contraindication to the use of tryparsamide in primary optic atrophy requires some revision. The incidence of serious visual damage [that is, the superposition of a tryparsamide reaction on preëxisting optic atrophy, or the hastening of visual failure from optic atrophy itself, is only about 10 times more frequent than in patients with previously normal eyes (about 35 as compared to 3.5 percent)]. It seems further clear that if such visual damage does not occur, tryparsamide may be a valuable drug in arresting visual failure in optic atrophy. Moore's 1932 statement may therefore properly be rephrased as follows: *tryparsamide is relatively contraindicated in the treatment of syphilitic primary optic atrophy.* Nevertheless, the cautious therapist will doubtless still prefer methods of treatment

(vide infra) which do not carry with them a one-in-three risk of further sudden visual impairment, and which have been much more thoroughly demonstrated than tryparsamide to be productive of arrest of the atrophic process.

The subdural treatment of optic atrophy

Moore's 1932 review[1] listed the outcome in 138 patients with optic atrophy treated subdurally by 11 different observers. It was pointed out that the favorable effect of subdural treatment in neurosyphilis was probably due to the aseptic meningitis produced by the injected substance (whether an arsphenamine dissolved in salt solution or spinal fluid, arsphenaminized or mercurialized serum, or air) rather than to direct treponemicidal effect.

Since 1932, only four authors have reported on the use of subdural treatment in optic atrophy. Their results are presented in table 1. The most outstanding exponent of the method is Schacherl,[32] who has treated 114 patients, 69 of whom he has been able to follow for periods ranging from 3 to 20 years (no details given, but many of his patients have been followed for 10 years or longer). Three of his patients had unilateral optic atrophy, and in them the uninvolved eye remained normal for 15 to 18 years.

A dramatic variation of the intraspinal method was tried by Suker and Jacobson.[33] In 32 patients, they trephined the skull and injected into a lateral ventricle 1/50 grain of bichloride of mercury mixed with a variable amount of previously withdrawn ventricular fluid. Four to six injections, at intervals of 10 to 14 days, were given. Four of the patients became blind and two died, presumably as a result of treatment. The results of this method are obviously no better than those from the simpler intraspinal subdural method and there is much more danger to life.

TABLE 1

THE RESULTS OF TREATMENT IN PRIMARY OPTIC ATROPHY TREATED SUBDURALLY

Author and Reference	Total Cases	Improved or Stationary	Worse or Blind
Assembled from literature by Moore,[1] omitting Schacherl—see below	133	71	62
Dragomir[31]	5	5 "favorable influence in all." No further details.	
Schacherl[32]	69	53 Many for periods longer than 10 years.	16
Suker and Jacobson[33]	21	13	8
Gross and Lehrfeld[34]	31	11	20
Total	259	153 (58.2 percent)	106

In spite of the paucity of recent reports and the inadequate documentation of all the authors quoted in this and the previous review, it seems clear that since 58 percent of the treated cases have been apparently arrested for periods ranging from 1 to 20 years, the method deserves still further study.

The fever treatment of primary optic atrophy: Malaria

In the 1932 review[1] it was possible to gather only 89 cases treated by means of malaria inoculata. In view of the importance of the subject, these cases to-

TABLE 2

RESULTS OF TREATMENT IN PRIMARY OPTIC ATROPHY TREATED WITH MALARIA

Author and Reference	Total Cases	Arrested or Improved		Worse
		Observed Longer than 1 year	Observed Less than 1 year	
Arruga[35]	6	2	2	2
Behr[36]	8		5 (duration obs. not stated)	3
Bering[37]	6	3		3
Busacca[38]	2			2
Carey[39]	3	2	1	
Chopra and Das Gupta[40]	2		2 (Obs. period too short for value)	
Clark[41]	12	8		4 (advanced)
Dreyfus and Hanau[42]	1			1
Ebaugh[43]	2	2		
Elschnig[44]	18	1	7	10 (all but one advanced)
Fischer-Ascher[45]	18	2	11	5 (advanced)
Fleischer[46]	8	7	1	
Gala[47]	8	opposes method	3 (Obs. period not stated)	5
Gasteiger[48]	21	6		15 (all advanced)
Goria[49]	1	1		
Grage[50]	6	3		3
Havel[51]	16		7 (Obs. period not stated)	9
Heinsius[52]	1	1 (8 years)		
Hessberg[53]	11	1	2	8
Horn and Kauders[54]	5	5		
Horn and Kogerer[55]	2	1	1	
Huhn[56]	6			6
Jaensch[57]	6			6
Jossmann[58]	4	4	1	
Lasalle, Sohier, and Aujaleu[59]	1			
Longo[60]	20	(cannot determine observation periods)	18	2
O'Leary and Walsh[61]	48	16		32
Paulian[62]	29	16		13
Pedercine[63]	2	2		
Pires[64]	2	2		
Sabbadini[65]	6	5		1
Teräskeli[66]	19	5		14
Weskamp[67]	16	13 (2–8 years)		3
Wile and Hand[68]	15	10		5
Wolff[69]	20	5	3	4
Total	343	123	64	156

gether with those since reported are gathered together in table 2, and now total 343. This does not represent the total number of patients with optic atrophy treated with malaria, since many are buried in reports which bear the gen-

eral title of "The treatment of neurosyphilis." It does, however, represent all those readily obtained by an exhaustive search of the literature on the treatment of tabes dorsalis or of primary optic atrophy, and is certainly a large enough number from which to draw conclusions.

Unfortunately, most of the papers cited suffer from the same defects discussed in 1932; namely, few of them supply ophthalmologic details before and after treatment, and the periods of observation after treatment are often short or undeterminable.

Nevertheless, of the 343 patients treated by this method, apparent arrest (sometimes improvement) was brought about in 54.5 percent, and for longer than one year in at least 35.9 percent. In some instances (notably Weskamp,[67] whose results as to visual acuity are separately presented in table 3), the observation periods ranged from 2 to 7 years; that is, improvement or arrest for much longer periods of time than might have been expected without treatment (vide supra).

In addition to the authors cited in the table, Artwisky and Ostrewski, Chaillous, and Margareth (quoted by Hambresin[70]), and Pensitur, Orzechowski, and Marinesco (quoted by Schiff-Wertheimer and l'Hermitte[71] (all of these probably from the discussions of the papers of others, since the original references cannot be located); and Fribourg-Blanc,[72] in a journal inaccessible to us, favor the use of malaria in primary optic atrophy.

It seems clear, from the many reports cited, that poor results (further progression to blindness) have for the most part occurred in patients with atrophy that was far advanced when treatment was begun. While it is not possible, be cause of inadequate data, to document the point, it seems further clear that when malaria treatment is given to patients with early optic atrophy—that is, visual acuity in the better eye 20/40 or better—the result is arrest or slight improvement in a much higher proportion than the 54 percent indicated by the table.

Many of the former opponents of malaria treatment (for example Behr, O'Leary, Gasteiger, Igersheimer) have apparently swung around to the point of

TABLE 3

WESKAMP'S MALARIA-TREATED CASES OF OPTIC ATROPHY[67]

Case	Vision Before		Vision After		Duration of Observation	Remarks
	O.D.	O.S.	O.D.	O.S.		
1	1/4	1/10	1/3	1/10	7 yrs.	Improved
2	2/3	1/2	1	1	5 yrs.	Worse
3	1/4	1/6	0	1/10	2 yrs.	Worse
4	1/4	1/3	1/3	1/2	6 yrs.	Improved
5	1/3	2/3	1	1	6 yrs.	Improved
6	1/2	2/3	2/3	2/3	1½ yrs.	Improved
7	1/3	1/4	1/3	1/6	1½ yrs.	Worse
8	2/3	1/3	1	1	2 yrs.	Improved
9	1/3	1/4	1/2	1/3	3 yrs.	Improved
10	1	2/3	1	1	5 yrs.	Improved
11	1/3	1/6	0	0	6 mos.	Worse
12	2/3	2/3−	1	1	2¼ yrs.	Improved
13	1/2	1−	1/2	1	3 yrs.	Stat.
14	1	2/3	1	1	4 yrs.	Improved
15	1	2/3	1	1	3½ yrs.	Improved
16	2/3	1/3	2/3	2/3	3½ yrs.	Improved

view that in certain early cases, at least, arrest may be secured by this means.

Much more exact definition of the results obtainable is, however, desirable, with full details as to ophthalmologic and neurologic findings and prolonged post-treatment observation.

Optic atrophy treated with fever produced by mechanical means

Since 1932, Busacca[73] (4 cases) and Prati[74] (1 case) have treated patients with optic atrophy by artificial fever produced by the intramuscular injection of sulfur, with reputed favorable results; but only Prati's patient was followed long enough (4 years) to justify this conclusion.

Fever produced by mechanical or electrical means has been used by Neymann and Osborne[75] (7 cases), Culler and Simpson[76] (16 cases), and Menagh[77] (10 cases—only 9 followed) in the treatment of syphilitic primary optic atrophy. Of the 32 treated and followed patients, arrest or slight improvement occurred in 22 (70 percent) over observation periods ranging from 10 to 20 months. While these results appear superior to those from malaria, it must be recalled that the number of patients treated with mechanical fever is so far very small, the majority of them had relatively early optic atrophy (as compared to the many nearly hopeless cases treated with malaria), and the observation periods are still too short for definite conclusions to be drawn.

Summary

We present, preliminary to the publication of our own detailed studies, a review of the current literature on the incidence, diagnosis, and the results of conventional forms of treatment (tryparsamide, subdural treatment, malaria, electropyrexia, or mechanical fever) of syphilitic primary optic atrophy.

References

[1] Moore, J. E. The syphilitic optic atrophies, with especial reference to primary optic atrophy, Medicine, 1932, v. 11, p. 263.
[2] Moore, J. E., and Woods, A. C. The pathology and pathogenesis of syphilitic primary optic atrophy. A critical review. Amer. Jour. Ophth., 1940, v. 23, Jan., p. 1.
[3] Zeeman, W. P. C. Atrophia nervi optici tabetica. Nederl. Tijdschr. v. Geneesk., 1933, v. 77, p. 5069.
[4] Comauer, A. F. Atrofies opticas—parte clinica. Rev. Asoc. Med. Argent., 1935, v. 49, p. 989.
[5] Schwarzenburg, C. Pathologie und Pathogenese der tabischen Sehnervenatrophie und über die Augensymptome bei der Tabes. Tung-Chi Med. Monatschr., 1938, v. 13, pp. 243, 292, and 309.
[6] Marinesco, G., Draganesco, S., and Grigoresco, D. Paraplégie syphilitique d'Erb avec atrophie des nerfs optiques. Rev. d'Oto-Neuro-Opht., 1936, v. 14, p. 8.
[7] Monterro Sales. Atrofia luetica dos nervos opticos sobre 23 casos. Arq. Inst. Penido Burnier, 1937, v. 4, p. 28.
[8] Jefferson, G. Compression of the chiasma, optic nerves, and optic tracts by intracranial aneurysms. Brain, 1937, v. 60, p. 444.
[9] Moore, J. I. An etiologic study of optic neuropathies. Amer. Jour. Ophth., 1937, v. 20, p. 1099.
[10] Sloan, L. L., and Woods, A. C. Perimetric studies in syphilitic optic neuropathies. Arch. of Ophth., 1938, v. 20, p. 201.
[11] Behr, C. Das Verhalten und die diagnostische Bedeutung der Dunkeladaptation bei den verschiedenen Erkrankungen des Sehnervensystems. Klin. M. f. Augenh., 1915, v. 55, p. 449.
[12] Igersheimer, J. Das Verhalten der Dunkeladaptation bei Erkrankungen der optischen Leitungsbahn. Arch. f. Ophth., 1919, v. 98, p. 67.
[13] Schindler, E. Ueber die klinische Bedeutung der Dunkeladaptation. Klin. M. f. Augenh., 1922, v. 68, p. 710.
[14] Gasteiger, H. Ueber Strörungen der Dunkeladaptation bei Sehnervenenerkrankungen und ihre diagnostische Verwertbarkeit. Klin. M. f. Augenh., 1927, v. 78, p. 827.

[15] Accardi, V. La soglia dell'eccitamento luminoso in condizioni normali e pathologiche e sotto l'azione di alcuni farmaci. Ann. di Ottal. e Clin. Ocul., 1933, v. 60, p. 561.
[16] Rutgers, G. E. Zur Dunkelanpassung. Klin. M. f. Augenh., 1923, v. 71, p. 449.
[17] Leibrecht. Untersuchungen über die Adaptation der gesamten Netzhaut im kranken Auge. Arch. f. Augenh., 1898, v. 36, p. 211.
[18] Lohmann, W. Untersuchungen über Adaptation und ihre Bedeutung für Erkrankungen des Augenhintergrundes. Arch. f. Ophth., 1906-7, v. 65, p. 365.
[19] Stargardt, K. Ueber Störungen der Dunkeladaptation. Arch. f. Ophth., 1909, v. 73, p. 77.
[20] Wölfflin, E. Ueber Dunkeladaptation von fovealen und parafovealen Netzhautpartien. Arch. f. Ophth., 1910, v. 76, p. 464.
[21] Rutgers, G. E. Die Dunkeladaptation bei einigen Augenkrankheiten. Klin. M. f. Augenh., 1923, v. 71, p. 589.
[22] Foroni, C. Die Ophthalmotonangioskopie in der Frühdiagnose der optischen Neuritis und der Anfangsatrophie des Nervus opticus. Arch. f. Ophth., 1936, v. 136, p. 100.
[23] La Croix, A. La nerf optique et les arsenicaux trivalents et pentavalents. Ann. d'Ocul., 1933, v. 170, p. 572.
[24] Berens, C. The eye and its diseases. Philadelphia, W. B. Saunders Co., 1936.
[25] Skirball, J. J., and Thurman, F. M. Ocular reactions due to arsphenamine. Amer. Jour. Syph. & Neurol., 1935, v. 19, p. 197.
[26] Sloan, L. L., and Woods, A. C. Effect of tryparsamide on the eye. Amer. Jour. Syph., Gonor. & Ven. Dis., 1936, v. 20, p. 583.
[27] Wagener, H. P. The effect of tryparsamide on the optic nerve. Amer. Jour. Med. Sci., 1937, v. 193, p. 286.
[28] Cady, L. D., and Alvis, B. Y. The use of tryparsamide in patients with and without ocular lesions. Jour. Amer. Med. Assoc., 1926, v. 86, p. 184.
[29] Lees, D. Treatment of primary optic atrophy with tryparsamide. Trans. Ophth. Soc. U. Kingdom, 1932, v. 52, p. 203.
[30] Mayer, L. L. Tryparsamide therapy of neurosyphilis and atrophy of the optic nerve. Jour. Amer. Med. Assoc., 1937, v. 109, p. 1793.
[31] Dragomir, T. Traitement de l'atrophie tabétique par des injections intrarachidiennes au serum salvarsanisée. Bull. Soc. Roum. Neur., 1933, v. 1, p. 35. Abstr. in Zentralbl. f. d. ges. Ophth., 1934, v. 30, p. 668.
[32] Schacherl, M. Zwanzig Jahre endolumbaler Salvarsanbehandlung der Atrophia nervi optici tabetica. Wien. Klin. Woch., 1937, v. 50, p. 313.
[33] Suker, G., and Jacobson, M. The management of tabetic optic atrophy. Illinois Med. Jour., 1933, v. 63, p. 21.
[34] Gross, E., and Lehrfeld, L. Personal communication.
[35] Arruga, H. Quoted by Schiff-Wertheimer and l'Hermitte.[71]
[36] Behr, C. Ueber die anatomische Grundlagen und über die Behandlung der tabischen Sehnervenatrophie. Münch. Med. Woch., 1926, v. 73, pp. 311 and 366.
———. Ueber Metalues des Auges. Zeit. f. Augenh., 1926, v. 60, p. 319.
[37] Bering, F. Die Behandlung der Tabes mit Malaria. Deutsche Med. Woch., 1926, v. 52, p. 1611.
[38] Busacca, A. Augenerkrankungen bei der Malariatherapie. Rev. Ophth. São Paulo, 1932, v. 2, p. 96. Abstr. in Zentralbl. f. d. ges. Ophth., 1933, v. 29, p. 221.
[39] Carey, R. Optic atrophy complicating neurosyphilis and its treatment. Med. Bull. Vet. Admin., 1935-6, v. 12, p. 308.
[40] Chopra, R. N., and Das Gupta, B. M. Preliminary note on the treatment of neurosyphilis with monkey malaria. Indian Med. Gaz., 1936, v. 71, p. 187.
[41] Clark, C. P. The effect of malaria on certain syphilitic conditions of the eye. Trans. Amer. Ophth. Soc., 1934, v. 32, p. 452.
———. Role of malaria in control of optic atrophy due to syphilis. Arch. of Ophth., 1936, v. 15, p. 250.
[42] Dreyfus, G. L., and Hanau, R. Fiebertherapie der Tabes. Deutsche Zeit. f. Nervenh., 1926, v. 94, p. 218.
[43] Ebaugh, F. G. Treatment of tabes and cerebral syphilis with malaria. Jour. Amer. Med. Assoc., 1928, v. 91, p. 1020.
[44] Elschnig, A. Therapie der tabischen Sehnervenatrophie. Klin. M. f. Augenh., 1925, v. 75, p. 773.
[45] Fischer-Ascher, M. Malariabehandlung der tabischen Sehnervenatrophie. Klin. M. f. Augenh., 1926, v. 76, p. 102.
[46] Fleischer, B. Zur Fieberbehandlung der tabischen Sehnervenatrophie. Klin. M. f. Augenh., 1933, v. 90, p. 335.

[47] Gala, A. Malariabehandlung der tabischen Sehnervenatrophie. Časopis lékařú českých., 1927, v. 66, p. 614. Abstr. in Zentralbl. f. d. ges. Ophth., 1927, v. 18, p. 799.
[48] Gasteiger, H. Zur Malariabehandlung der tabischen Sehnervenatrophie. Arch. f. Augenh., 1934, v. 108, p. 471.
[49] Goria, C. Sui resultati della terapie colla malaria e la ricorrente nella tabe. Minerva Méd., 1927, v. 7, p. 494.
[50] Grage. Die Fieberbehandlung bei tabischer Sehnervenatrophie. Deutsche Zeit. f. Nervenh., 1932, v. 127, p. 74.
[51] Havel, J. Malariabehandlung der tabischen Sehnervenatrophie. Časopis lékařú českých., 1928, v. 67, p. 1055. Abstr. in Zentralbl. f. d. ges. Ophth., 1929, v. 20, p. 610.
[52] Heinsius, E. Zur Frage der Malariabehandlung der tabischen Optikusatrophie. Zeit. f. Augenh., 1935, v. 87, p. 298.
[53] Hessberg, R. Weitere Ergebnisse der Fieberbehandlung luetischen insbesondere tabischen Sehnervenerkrankungen. Klin. M. f. Augenh., 1930, v. 84, p. 261.
[54] Horn, L., and Kauders, O. Eine neue Methode der abgeschwächten Malariabehandlung. Wiener Klin. Woch., 1928, v. 41, p. 585.
[55] Horn, L., and Kogerer, H. Ueber die Ergebnisse der Nachuntersuchung der mit Lufteinblasung behandelten Fälle von tabischer Optikusatrophie. Zeit. f. Augenh., 1929, v. 67, p. 207.
[56] Huhn, K. Tabische Atrophie der Sehnerven und ihre Therapie durch Malaria. Klin. M. f. Augenh., 1928, v. 80, p. 693.
[57] Jaensch, P. A. Zur Therapie des tabischen Sehnervenschwundes. Zeit. f. Augenh., 1930, v. 71, p. 12.
[58] Jossmann, P. Erfolge der Reiztherapie bei progressiver Paralyse. Allg. Zeit. f. Psychiat., 1931, v. 95, p. 321.
[59] Lassale, Sohier, R., and Aujaleu, E. Atrophie optique tabétique et malariathérapie. Soc. de Méd. Mil. Franç., 1934, v. 28, p. 288.
[60] Longo, P. W. Resultado da malario terapia na atrofia do nervo optico devida a lues: Estudo de 20 casos. Arq. Inst. Penido Burnier, 1936, v. 4, p. 183.
[61] O'Leary, P., and Walsh, A. L. Treatment of primary optic atrophy with malaria. Jour. Amer. Med. Assoc., 1933, v. 101, p. 493.
[62] Paulian, D. Le traitement du tabès par la malariathérapie. Paris Méd., 1928, v. 1, p. 231.
[63] Pedercine, E. Atrofia specifica dell'ottico e terapia malarica. Osp. Bergamo, 1933, v. 4, p. 183. Abstr. in Zentralbl. f. d. ges. Ophth., 1936, v. 35, p. 309.
[64] Pires, W. Tabes juvenil. An. da assist. a psicopat., 1931, p. 29.
[65] Sabbadini, D. Osservazioni a distanza atrofie ottiche tabetiche sottoposte alla malaria terapia. Riv. Oto-Neuro-Oftal., 1933, v. 10, p. 43.
[66] Teräskeli, H. Malariatherapie bei Sehnervenschwund infolge Tabes und Paralyse. Duodecim, 1936, v. 52, p. 1000. Abstr. in Zentralbl. f. d. ges. Ophth., 1937, v. 38, p. 441.
[67] Weskamp, C. Atrophies optiques et malariathérapie. Ann. d'Ocul., 1936, v. 172, p. 499.
[68] Wile, U. J., and Hand, E. A. Ten years' experience with malaria in neurosyphilis. Amer. Jour. Syph., Gonor., & Ven. Dis., 1936, v. 20, p. 630.
[69] Wolff, J. Behandlung der Atrophia nervi optici durch Malariakuren und Pyriferinjektionen. Abstr. in Zentralbl. f. d. ges. Ophth., 1930, v. 22, p. 812.
[70] Hambresin, L. Pyretotherapy in ophthalmology. Arch. of Ophth., 1938, v. 19, p. 497.
[71] Schiff-Wertheimer, S., and l'Hermitte, J. Pathogénèse et traitement de l'atrophie optique tabétique. Bull. Soc. Ophth. de Paris, 1932, v. 44, pp. I-XCV.
[72] Fribourg-Blanc, A. La malaria thérapie de l'atrophie optique. Rev. Méd. Franç., 1934, v. 15, p. 511.
[73] Busacca, A. Schwefeltherapie bei luetischer Atrophia nervi optici und anderen Augenkrankheiten. Klin. M. f. Augenh., 1933, v. 90, p. 352.
[74] Prati, L. Su di un caso di atrofia ottica luetica trattato con le piretoterapia. Forte e stobile miglioramento. Riv. Oto-Neuro-Oftal., 1935, v. 12, p. 286.
[75] Neymann, C. A., and Osborne, S. L. The development of hyperpyrexia. Arch. Phys. Therapy, 1934, v. 15, p. 149.
Neymann, C. A. Artificial fever produced by physical means: Its development and application. Springfield, Ill., Charles C Thomas, 1938, p. 149.
[76] Culler, A., and Simpson, W. Artificial fever therapy in cases of ocular syphilis. Amer. Jour. Ophth., 1936, v. 15, p. 624.
[77] Menagh, F. R. Treatment of syphilis with hyperpyrexia with observations on the prognosis of optic atrophy. Amer. Jour. Syph., Gonor. & Ven. Dis., 1937, v. 21, p. 609.

METASTATIC MALIGNANT MELANOMA OF THE RETINA*

ELLA M. UHLER, M.D.
Baltimore

Metastatic involvement of the retina by carcinoma or sarcoma is rare. The volumes of Lagrange,[1] Henke-Lubarsch,[2] and Morax[3] do not mention metastatic involvement of the retina. Sattler[4] records three cases, those of Schiess-Gemuseus and Roth, of Heine, and of Arisawa, all of which involved both the optic disc and the retina. Two other cases have been reported, both of which involved the retina alone: those of Boente,[5] and of Smoleroff and Agatston.[6] Of these five cases of retinal metastasis, two were cases of malignant melanoma, one was of round-cell sarcoma, and two were of carcinoma.

The first case is that recorded by Schiess-Gemuseus and Roth[7] in 1879 in a 40-year-old man who, six months after the removal of a large mole on the sternum, had metastases to the right axilla and simultaneously noticed dimness of vision in the left eye. Four months later all vision was lost in this eye. The eye was enucleated. One year after the first evidence of ocular involvement the patient died of general metastases. The primary tumor as well as the metastases was a pigmented spindle-cell sarcoma with sparse intercellular substance.

Sagittal section of the left eye showed a small mushroomlike tumor projecting into the vitreous from the head of the optic nerve. The main portion of the tumor did not extend behind the anterior border of the lamina cribrosa, but a few fingerlike projections penetrated back into the lamina parallel to the optic-nerve fibers. The retina immediately adjacent to the optic nerve was replaced by the tumor, which soon ended abruptly in the intact layers of the adjoining retina. The tumor did not penetrate the choroid. It consisted chiefly of short pigmented spindle cells with delicate reticular stroma. The spindle cells were thickly aligned along the walls of the numerous blood vessels penetrating the tumor.

The second case, recorded by Heine,[8] occurred in a 50-year-old patient. A large ulcerated tumor was removed from the interscapular region three months after its appearance. Soon after this operation there were metastases in both axillae, and two months later the patient died. On autopsy numerous metastases were found in the lungs.

There was no loss of vision, but shortly before death a small, reddish, well-circumscribed tumor was seen occupying the greater part of the optic disc and the immediately adjoining part of the nasal half of the retina. A sickle-shaped portion of the optic disc was still recognizable temporally. The tumor extended with a peglike projection surrounding an arteriole to the lamina cribrosa, and was traversed by several apparently intact nerve-fiber bundles. The retinal metastasis had the structure of a round-cell sarcoma, as did the primary tumor and the lung metastases.

Boente's[5] patient was a 45-year-old man who gave a history of the removal of a primary pigmented tumor over the right parietal bone. When first seen, he had generalized subcutaneous metastases. One month after admission, the patient complained of flickering in the right eye. In the retina of the right eye were two small tumor nodules in the temporal part

* From The Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

of the globe, half way between the ciliary body and the posterior pole. Both sat upon large veins without occluding their lumina. One month later there was considerable diminution in vision; other melanoblastic nodules had now appeared in the retina. The patient died three months after the first evidence of ocular involvement.

Autopsy showed generalized metastases of melanosarcoma. The choroid of the right eye contained innumerable metastases but nowhere was the lamina vitrea or the pigment epithelium invaded. Eight separate metastases were found in the retina. These lay in the nerve-fiber layer, in the walls or lumen of retinal veins. The tumor consisted chiefly of unpigmented spindle cells, with a smaller number of large pigmented round cells. The optic nerve contained an isolated metastasis 0.9 cm. from the disc.

The first case of metastatic carcinoma of the retina was reported by Arisawa[9] in a 30-year-old patient who at laparotomy was found to have an extensive inoperable carcinoma, the primary site of which appeared to be the pancreas. Four months later he complained of dimness of vision in the right eye, and a tumor was seen projecting far forward into the vitreous. Four months afterward the eye was enucleated because of severe pain. The patient died 12 days after this operation. No autopsy was obtained.

A partially cystic tumor, connected by a pedicle to the head of the optic nerve, filled two thirds of the vitreous. The tumor was composed of cuboidal epithelium in partial alveolar structure, and penetrated somewhat into the optic nerve, partially destroying the lamina cribrosa. Most of the nerve fibers were degenerated. The greater part of the retina was replaced by the tumor, but the latter bore no relation to the choroid. There was no metastasis in the stalk of the optic nerve.

Smoleroff and Agatston[6] reported a metastasis of a gastro-esophageal carcinoma involving only the retina proper. The patient, a 55-year-old man, had no complaints referable to the eyes. On ophthalmological examination a white, irregular, elevated mass was seen in the lower temporal quadrant of the fundus of the right eye. A month after admission the patient died. Autopsy showed generalized carcinoma of the cardiac end of the stomach with metastasis.

On horizontal section there was seen a uniform white mass in the temporal portion of the right globe, elevating the retina. Microscopic examination showed that the retina was detached from a point very near the ora serrata to the region of the optic nerve by exudate and a tumor. The latter, which had the appearance of an alveolar adenocarcinoma, arose from the nerve-fiber layer, one group of cells appearing to spring from the wall of a blood vessel. The tumor destroyed the various layers of the retina and broke through the pigment epithelium into the subretinal space. The lamina vitrea was intact. There was no involvement of the choroid nor of any other part of the globe.

Case report

The case to be reported is that of metastasis of a malignant melanoma to the retina without optic disc or choroidal involvement.

W. K., a 26-year-old white man, was admitted to the surgical service of the Johns Hopkins Hospital on November 14, 1926, complaining of weakness of the left leg, headaches, and vomiting. The family history and past history were noncontributory.

Six weeks before admission he began having pain in the shoulders, malaise,

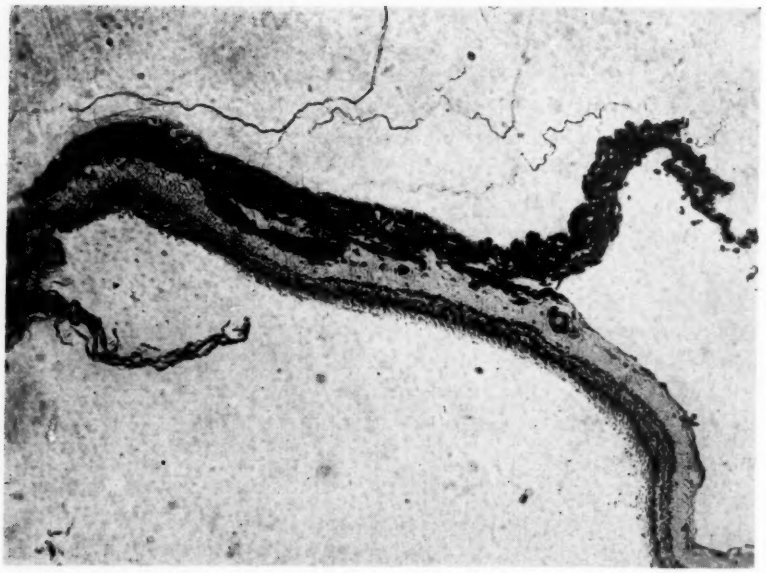


Fig. 1 (Uhler). Photomicrograph showing full extent of metastasis (×100).

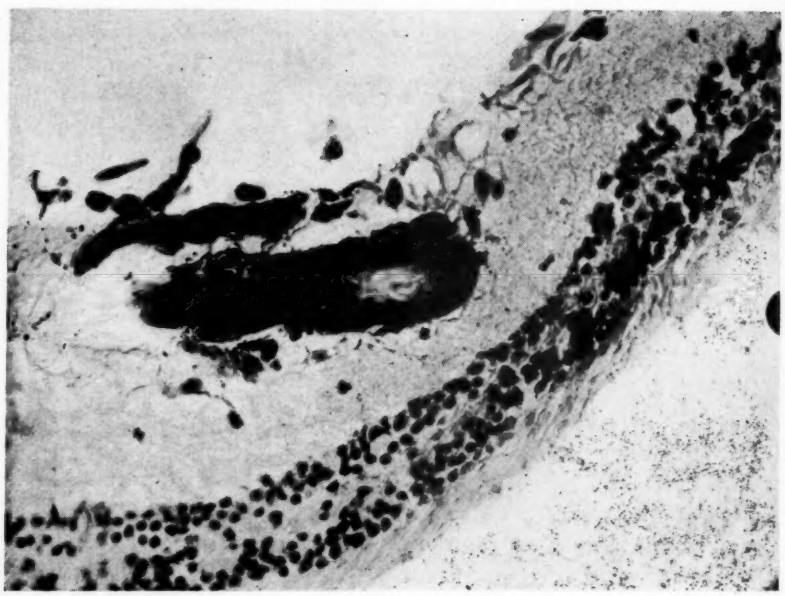


Fig. 2 (Uhler). Photomicrograph showing perivascular arrangement of tumor cells (×450).

soreness of both lower extremities, and weakness of the left leg. Three weeks before admission the patient had severe continuous headaches with vomiting. He complained of diplopia just before admission.

General physical examination showed weakness of the right sixth nerve, paralysis of the right seventh nerve, and absence of deep reflexes.

Ophthalmological examination showed blurring of both discs, no other abnormalities.

Course in the hospital: On November 28, 1926, pigmented nodules appeared in the patient's chest. Biopsy of one of these

nodules was described as showing melanotic sarcoma. During his stay in the hospital there was a gradual loss of weight, a progressive paralysis of both fifth, seventh, and eighth nerves, as well as progressive formation of subcutaneous nodules. He died on January 18, 1927. Clinical impression: Diffuse melanotic sarcomatosis.

General autopsy report. (Dr. M. K. Smith): The anatomical diagnosis of the general autopsy was: melanotic sarcoma, origin undetermined. Tumor nodules occurred in the skin and subcutaneous tissue, lymph nodes, brain, skull, intestine, adrenal, and lung. Tumor nodules involved the cranial nerves, the spinal cord, and cauda equina in the lower lumbar region. The tumor tissue consisted of bundles of spindle-shaped cells which often contained finely divided brown pigment. Large oval phagocytic cells containing coarse pigment granules lay scattered throughout the tumor nodules.

Pathologic examination of the eyes

Left eye: The globe was of normal size and shape. The cornea was diffusely opaque. The center of the corneal surface was denuded of epithelium, and between the anterior lamellae were thick colonies of bacteria accompanied by a diffuse polymorphonuclear exudate. There was minimal infiltration of the iris. A moderate choked disc was present. No other abnormalities were seen. There was no evidence of metastasis.

Right eye: Only the posterior half of the globe was available for examination. This was of normal size and shape. The vitreous appeared normal and the retina and choroid were in place. There was marked choked disc.

Serial horizontal sections showed a band of tumor cells in the temporal portion of the retina, extending from the ora serrata toward the posterior pole for a distance of about 2 mm., replacing the nerve-fiber and ganglion-cell layers. The growth was about 1 mm. in width and about 0.1 mm. in height. There was no invasion of the vitreous, the internal limiting membrane being intact over the tumor. Scattered small vessels in the inner plexiform layer of the retina below the main tumor were ensheathed by concentric layers of the tumor cells. The sheaths of the vessels nearest the ganglion-cell layer were continuous with the main tumor mass. The internal nuclear layer and the succeeding outer layers of the retina were not invaded. The lamina vitrea was intact. The tumor ended sharply at the ora serrata. No tumor cells were present in the pars plana of the ciliary body. There was no metastasis in the choroid.

The tumor consisted chiefly of dense parallel strands of spindle-shaped cells with elongated oval nuclei containing a delicate reticular structure with prominent nucleoli. The cytoplasm was scant and contained abundant finely divided brown pigment. The cells ended in fibrillary processes which appeared to be continuous with those of the adjoining cells. At the edges of the main tumor mass were occasional large polygonal cells densely packed with coarse brown pigment. This combination of spindle and epithelioid cells was similar to that described by Callendar,[10] as the mixed-cell type of the primary malignant uveal neoplasms.

Comment

As far as can be found in the literature, the case reported is the first in which metastatic involvement of the retina by a malignant melanoma occurred without involving the optic disc or choroid.

There are six reported cases of metastasis to the retina, three of them being malignant melanoma, one round-cell sar-

coma, and two carcinoma. This is in marked contrast to the relatively far larger number of reported choroidal metastases. Ask[11] in 1934, tabulated 145 cases of choroidal metastasis of carcinoma proved by histological study. Lemoine and McLeod[12] reported the total number of proved cases in 1936 as 156. Fry,[13] in 1933, tabulated seven cases of metastatic sarcoma of the choroid. Thus there are in the literature a total of 163 cases of choroidal metastasis compared to six cases of retinal metastasis. There has been an overwhelming proportion of carcinoma in the choroidal metastasis in contrast to the retinal metastases in which malignant melanoma predominate, although the total number of retinal metastases is too small to give the comparison statistical significance.

Conclusions

The literature on metastatic involvement of the retina is reviewed.

A case is reported of metastatic involvement of the retina by a malignant melanoma without involvement of the optic disc or choroid.

References

[1] Lagrange, F. Traité des tumeurs de l'oeil. Paris, 1901.
[2] Henke, F., and Lubarsch, O. Handbuch der speziellen pathologischen Anatomie und Histologie. Berlin, 1928, v. 11, Aug., pt. 1.
[3] Morax, V. Cancer de l'appareil visuel. Paris, 1926.
[4] Sattler. Die bösartigen Geschwülste des Auges. Leipzig, 1926, p. 219.
[5] Boente. Metastatische Melanoblastome in der Retina. Klin. M. f. Augenh., 1929, v. 82, June 20, pp. 732-740.
[6] Smoleroff, J. W., and Agatston, S. A. Metastatic carcinoma of the retina. Arch. of Ophth., 1934, v. 12, Sept., pp. 359-365.
[7] Schiess-Gemuseus and Roth. Metastatisches Sarcom der Papille und angrenzenden Retina. Graefe's Arch. f. Ophth., 1879, v. 25, pp. 177-190.
[8] Heine, L. As quoted by Sattler,[4] op. cit., p. 219.
[9] Arisawa. Ueber echtes Carcinom der Retina. Klin. M. f. Augenh., 1914, v. 52, p. 386.
[10] Callendar. Malignant melanotic tumors of the eye. Trans. Amer. Acad. Ophth. and Otol., 1931, p. 131.
[11] Ask, O. Zur Kenntnis des metastatischen Uvealkarzinoms. Acta Ophth., 1934, v. 12, p. 308.
[12] Lemoine, A. N., and McLeod, J. Bilateral metastatic carcinoma of the choroid. Arch. of Ophth., 1936, v. 16, Nov., p. 804-821.
[13] Fry, W. E. Metastatic sarcoma of the choroid. Arch. of Ophth., 1933, v. 9, Feb., p. 249.

ESSENTIAL ATROPHY OF THE IRIS

With pathological report

Benjamin Rones, M.D.
Washington, D.C.

The clinical entity of essential atrophy of the iris, resulting in glaucoma, has been known since the beginning of the century. The underlying factors, however, are as little known now as then. Clinical reports are not abundant, and histologic examinations are even sparser. From the etiological viewpoint it is regrettable that all of the sectioned eyes showed advanced stages of glaucoma, and therefore offered no clue as to the beginning stages of hole formation in the iris. Controversy has revolved around the factor of the glaucoma production, with several theories offered which are not amenable to proof. Since only six microscopic examinations of such eyes are found in the literature, it was felt advisable to report the following case.

Case report

Mrs. B. S., aged 53 years, was first seen on September 7, 1938. In 1921 she had noticed that the pupil of the right eye was enlarged. It later became vertically oval in shape. There was no pain in the eye and vision was good. About 1928 she observed that the pupil had "separated into three parts, large, medium, and small." The eye was comfortable until April, 1938, when she began to have attacks of pain. At first these were of short duration and at fairly long intervals. Later the pain became more prolonged and of increased frequency, and the eye was markedly reddened during these attacks.

Examination of the eyes resulted in the following observations:

Right eye: The eye was white. A macula was present on the temporal side of the cornea at about the 8-o'clock position. With the slitlamp this was seen to be due to an irregularity of the epithelium and haziness in the superficial layers of the stroma. The posterior corneal surface was entirely clear, with no pigment granules nor cellular deposits. The iris appeared as in the diagram, with the pupil

Fig. 1 (Rones). Pupil with intact sphincter, and with two holes above and temporal to it. Nasal margins of holes show ectropion uveae.

displaced down and in, and the sphincter intact. The pupil did not, however, react to light. Above and temporal to the pupil two holes were present, reaching to the chamber angle on the temporal side. On the nasal margin of each hole there was an ectropion of the pigment layer of the iris. The lens was clear, both on ophthalmoscopic examination and under the slitlamp. A red reflex could be obtained from the pupil and from each of the holes. The nerve head showed deep glaucomatous cupping. At this time the vision was re-

duced to light perception. Tension to fingers was not elevated.

The left eye was normal in all respects.

The patient was informed that if the eye became painful it would be advisable to remove it. During the course of the next few months the pain became very marked, so that she consented to enucleation, which was performed on January 17, 1939.

Pathological report

The eye was sectioned serially, and every tenth section was mounted.

Many of the mesothelial cells seemed to contain melanin pigment. The iris was atrophic and markedly deformed. On one side an atrophic stub was completely adherent to the cornea and represented for quite a distance only by a layer of pigment epithelium (figs. 2 and 3). On the opposite side the iris remnant was thickened, due to its being doubled over itself as the result of synechia formation. This produced the ectropion uveae (figs. 2 and 7). Although a few round cells were scattered through the iris stroma there was no evidence of a previous inflammatory

Fig. 2 (Rones). Temporal root of iris is firmly adherent to posterior surface of cornea, while nasal root shows ectropion of pigment layer. Two holes are visible in this section, with central portion, which is magnified in figure 6.

Gross: There was a small raised white patch on the cornea between its center and the limbus. Three holes were present in the iris, the largest of which measured approximately 9 by 6 mm. and extended almost to the limbus on either side. At one end this was flanked by the two smaller holes which were 6 by 2 and 3 by 1 mm., respectively.

Microscopic: The corneal epithelium was slightly irregular. A few neutrophiles and round cells infiltrated the interlamellar spaces of the corneal stroma. Perivascular round cells were present in the conjunctival stroma. Peripheral anterior synechiae blocked the chamber angle. lesion, as is seen in figures 3, 4, and 5, which represent sections along one of the holes. The central portion of the iris (figs. 2 and 6) showed an area of loose stroma cells, while adjacent to this was a zone of densely packed fibrous-tissue cells. The stromal vessels were sclerotic and their lumina almost occluded. There were no posterior synechiae to be seen. The ciliary processes were moderately swollen and showed lipohyaline degeneration. The ciliary musculature was atrophic. Calcified drusen appeared along with lipohyaline drusen. Focal cystoid degeneration was seen in one area of the equator in the temporal area of the retina. The

Fig. 3

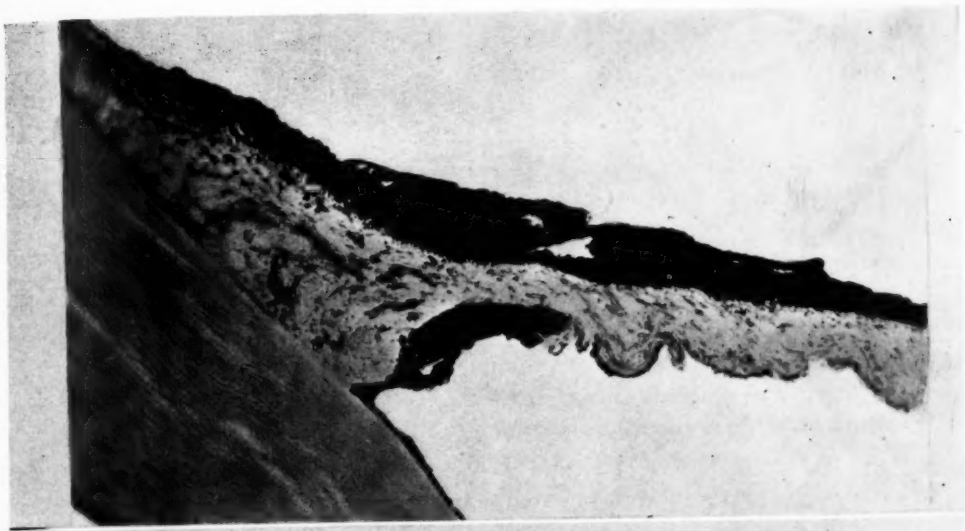


Fig. 4

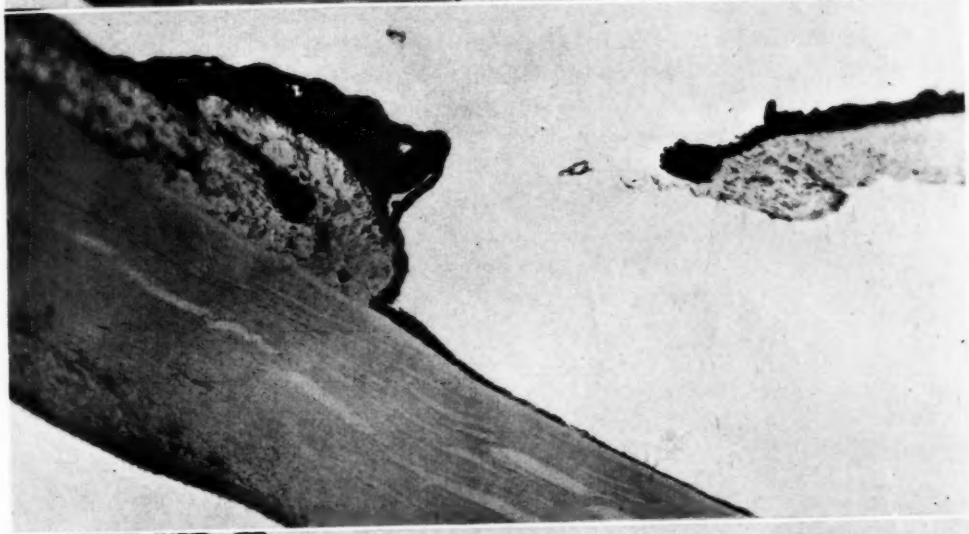


Fig. 5

Figs. 3, 4, and 5 (Rones). Sections along the same hole as in figure 2. Iris tissue presents no evidence of cellular reaction from inflammation.

Fig. 6 (Rones). Iris tissue between holes. One portion is composed of loose, atrophic connective tissue, while adjacent to it there are tightly packed fibrous-tissue cells in which blood vessels are scarce.

arteries of the nerve-fiber layer showed cellular thickening, which may be intimal. The capillaries in the macular region showed slight hyaline walls without marked decrease in the caliber of the lumina. The ganglion cells were markedly decreased in number. There was atherosclerosis of the choroidal arteries, which in places showed foam cells in the intima. The nerve head was markedly cupped and atrophic. Corpora arenacea were found in the dura mater of the nerve sheath.

Diagnosis: Chronic glaucoma, shallow-angle type. Irregular colobomatous atrophy of the iris. Clinical essential atrophy of the iris.

Discussion

The earliest cases reported in the literature do not seem to fall definitely into this class of "essential atrophy." Johnson[1] (1886) reported a patient, one of whose eyes had been lost as a result of an accident 18 years before. The other eye then developed an atrophy of the iris which progressed, so that only a narrow edge remained above and below. Johnson did not state whether any signs of inflammation were present, nor did he make any mention of the tension of the eye, although he did note the fact that the disc was normal.

Hess[2] (1892) contributed the report of a 14-year-old boy who developed poor vision after an attack of scarlet fever at the age of eight years. Examination showed the pupils to be eccentric and pushed temporally, and on the nasal side there was a large defect of the iris in each eye. The tension was markedly elevated in both eyes, and there was deep cupping of the discs. Because of the presence of small white points on the anterior lens capsule, Hess thought the condition to be due to a chronic iridocyclitis.

Harms[3] (1903) noted a case in which 10 years previously there had been noticed a small hole in the iris adjacent to the pupil. Later another hole occurred, and both slowly increased in size. Numerous precipitates were present on the posterior corneal surface, and in his opinion the cause was a chronic iridocyclitis.

Wood's[4] patient (1910) had a progressive tissue defect of the iris, so that only the pigment layer remained. The tension was elevated. The eye was enucleated, and on examination was found to have a chronic iridocyclitis, with extensive iris atrophy and degeneration, and a secondary glaucoma.

In the case of Lane[5] (1917) a hole developed at the margin of the pupil, and shortly afterwards another one developed

temporally; gradually other portions of the iris began to disappear, leaving only some small bands of tissue. The eye was painful and the tension elevated. There had been, however, several attacks of inflammation.

In all of these cases there were evidences of inflammatory disturbances in the eyes, either in the history or on examination, and consequently I am not convinced that these belong to the group of true "essential atrophies." The introduction of the corneal microscope enabled observers to detect early evidences of inflammation and thus made the differentiation clear-cut.

Feingold[6] (1918) presented a true case with no evidence of inflammation. Numerous holes were present in the ciliary portion of the iris, and the pupil was eccentric. The tension was elevated, and the pain persisted even under miotics. The eye was removed and sectioned serially. Adhesions of the iris root to the cornea were found, obliterating the chamber angle. The defects of the iris were of different degrees, some being actual holes, while others involved only the stroma and left the pigment layer intact. There were no evidences of inflammation either as cells on the cornea or as posterior synechiae. The blood vessels of the iris were numerous and some were thickened, although their lumina were not narrowed. The disc showed glaucomatous cupping. Feingold noted that these vascular changes involved only the ciliary portion of the iris but were absent in the pupillary portion where the sphincter muscle was normal.

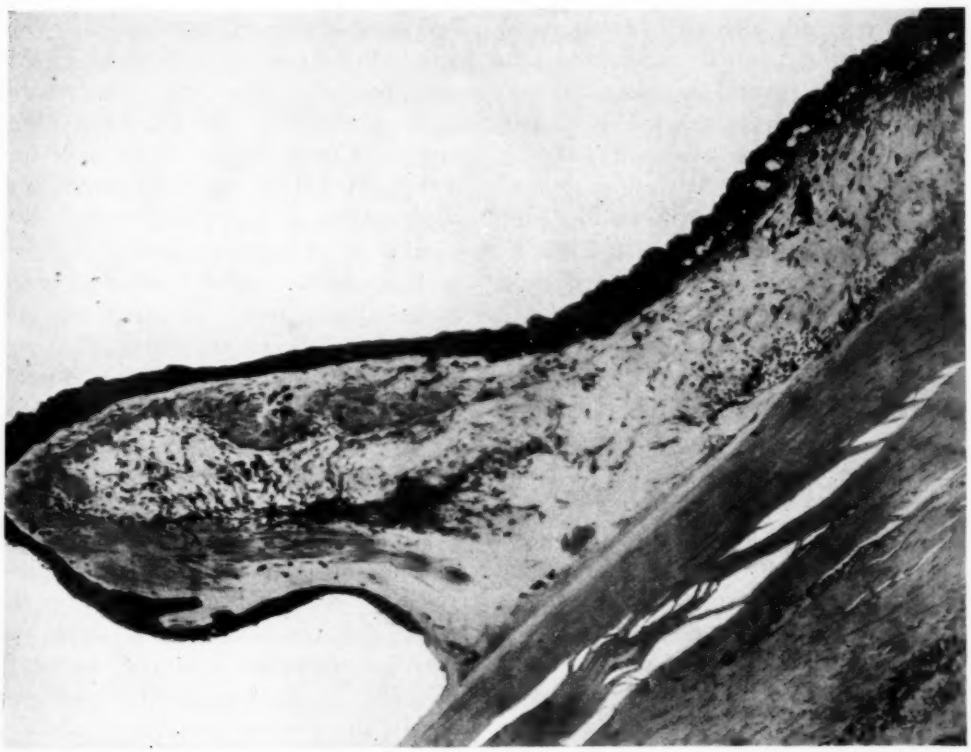


Fig. 7 (Rones). Temporal root of iris showing that ectropion is result of contracted adhesion to cornea. Dense fibrous tissue is prominent in stump. Stromal vessels show marked sclerosis.

Larsson[7] (1920) offered a case in which the pupil was pulled down and vertically, and there were numerous defects in the iris with some ectropion uveae; the tension was markedly elevated.

Licskó[8] (1923) reported data on a patient who remained under observation during the entire course of 15 years. The patient was first seen in 1917 by Pórs, who found two large holes temporal to the pupil. The vision was normal, and there was no mention of increased tension. By 1920 the tension had become elevated, so that an anterior sclerotomy was performed. The tension remained elevated, however, and there was poor light perception. The pupil was displaced nasally, and there was a large hole above and a small one nasal to it. In this case it is noteworthy that the blood Wassermann test was positive. The eye was enucleated and histologic examination showed pigment granules and clumps on the posterior corneal surface. The iris tissue was markedly thinned, very deficient in cells, and the stroma was atrophic. Clumps of pigment cells were present in the stroma and especially around the blood vessels, which showed hyaline degeneration. The iris was covered by a glassy membrane, and although its root touched the cornea it did not appear grown to it. The pigment epithelium was thickened and many of the cells were swollen. There was no evidence of inflammation.

In 1923 Arnold[9] described a case in which a hole had been noticed in the iris two years previously. He found the pupil to be pulled upward and inwards, its sphincter intact. A large hole was present beneath the pupil. There were no evidences of inflammation. The tension in this eye was normal, as was the vision. Arnold stressed the point that this was one of the few cases that had been reported in which there was no glaucoma. However, in 1936 Rieth[10] had the opportunity of reporting this same case. He stated that in 1924 the tension had become elevated in the eye, and had remained so even after pilocarpine therapy. The eye went on to the development of phthisis.

The case reported by Rochat and Mulder[11] (1924) is of considerable interest. In 1917 the pupil was found to be displaced temporally and a little upwards, and a hole was present on the nasal side of the iris. The vision, fields, and tension were normal. During the next six years the iris disappeared almost entirely. The tension became markedly elevated. Vision was abolished, and the disc showed deep cupping. The eye was removed and sectioned. The iris was adherent to the cornea in its entire circumference. These anterior synechiae were most markedly developed in the region where the pupil was pulled over to the limbus. In this zone the iris tissue was not rarefied, as in other places, but on the contrary was more compact. A new tissue had formed in the chamber angle containing many oblong cells whose nuclei were parallel to the corneal layers. The iris surface was covered with a hyaline membrane. There were no signs of preexisting inflammation.

Gifford's[12] case (1926) is of interest in that it was seen in 1922 with normal vision. During two years the pupil was drawn up and in, and at the lower part of the iris there was a sector of atrophy of the stroma with an intact pigment epithelium. This gradually progressed until a hole developed. The tension became elevated and required several trephining operations to keep it within normal limits.

In 1926 de Schweinitz[13] thoroughly reviewed the literature, and also added a case that he had observed since 1913. During its initial stages the vision and tension were normal, but with the evolu-

tion of the iris atrophy the tension became elevated and persisted in that state until vision was abolished.

Griscom[14] (1927) reported a case in which there was a hole on the temporal side, leaving the sphincter intact, and another hole at the ciliary border. There was no evidence of inflammation nor migration of pigment from the iris.

Ellett[15] (1928) presented a patient whom he had first seen in 1921, with the story that the pupil had been irregular for five years. He found the pupil to be pulled up and out, and the pillars attached to the cornea. During the next four years three holes developed in the lower part of the iris. Later there appeared a bullous keratitis and glaucoma which indicated enucleation. Examination of the sectioned eye showed the iris to be widely adherent to the cornea. This adherent iris was found to be markedly fibrosed and the free portion of the pupillary margin had an ectropion of the uveal layer. One portion of the iris showed a disappearance of the stroma, leaving only the dilator muscle and the pigment epithelium. The sphincter muscle was found to be intact.

Waite[16] (1928) reported a case which he states was one of three seen at the Massachusetts Eye and Ear Hospital. The patient was first seen in 1923. At this time the pupil was found to be drawn down and nasally, and there was beginning atrophy of the iris root above. The tension, visual fields, and optic nerve were normal. During the course of the same year the patient developed pain in the eye from the glaucoma, so that an operation was performed. The control of the glaucoma had no beneficial effect on the progressive atrophy of the iris. There was never any evidence of inflammation.

Jeancon's[17] patient (1933) had three large radial holes and an elevated tension. At the time that Barr[18] (1934) reported his case, it had gone on for six years, and two large holes had developed, but yet the fundus and the fields were normal.

Von Grosz[19] (1936) reported two definite cases, in one of which the tension was markedly elevated, and in the other was within normal limits. Neither showed any evidence of inflammation.

McKeown[20] (1937) presented the only case that has had a gonioscopic examination to date. This revealed wide anterior peripheral synechiae.

Fine and Barkan[21] (1937) presented a nine-year old boy, in whom both eyes were involved. An advanced stage of iris atrophy had developed in this patient at five years of age and was accompanied by glaucoma. The conditions went on to buphthalmos. The rarity of this case lies in its bilateral involvement; it is extremely doubtful whether it should be included within the true essential atrophies, but seems much more likely to be one of congenital glaucoma with a secondary iris atrophy.

Clinical course

From a study of these case records, one can determine a fairly definite progression of the symptoms. The initial sign is the displacement of the pupil, which also tends to become distorted. There then develops an atrophy of the stroma, usually in the portion of the iris opposite the direction of displacement of the pupil. This atrophy may be of varying extent, and the stroma gradually disappears leaving the pigment-epithelium layer exposed. This layer apparently has a greater resistance, but sooner or later it also becomes involved and complete holes are then visible through the thickness of the iris. The final symptom is the appearance of the glaucoma, although in Fine and Barkan's case the iris atrophy and glaucoma were concurrent.

Many theories have been offered as to the etiology of these symptoms. Microscopic examinations have been carried out by Bentzen and Leber, Licskó, Rochat and Mulder, Feingold, Ellett, and in the above-reported case. With the exception of Casey Wood's doubtful case, none showed any signs of inflammatory disease. In all of them the periphery of the iris was generally adherent to the cornea, and the still-existing portions of the iris were atrophic. Licskó and Feingold found some hyaline degeneration of the iris vessels. Feingold offered the theory that the atrophy of the iris was produced by a congenital vascular disturbance of the smaller iris circle. He suggested that the glaucoma was produced by irritating substances which were elaborated as a result of the destruction of the iris tissue. Licskó attributed the glaucoma partly to the dissemination of the pigment from the atrophic iris, and partly also to the atrophy of the iris tissue by which the surface available for resorption of intraocular fluid was considerably diminished.

Bentzen and Leber[22] believed that the glaucoma itself was the cause of the iris atrophy. This, however, is disproved by the numerous cases in which the iris atrophy definitely preceded the rise of tension.

Rochat and Mulder considered the principal factor to be the soldering together of the iris root to the periphery of the cornea, beginning at a circumscribed spot and leading to the displacement of the pupil. This pulling of the iris causes distention of the anterior layers of the opposite side, resulting in atrophy and finally tearing of the stroma and the pigment layer. The slowly progressing obliteration of the chamber angle finally blocks off the aqueous drainage and produces glaucoma.

Kreiker[23] assumed that the cytolitic process, which normally operates in embryonic life, causing the resorption of the pupillary membrane, becomes active in adult life and attacks the normal iris tissue. His view is that the glaucoma is produced by the cellular detritus suspended in the anterior chamber, causing an occlusion of the chamber angle.

Waite has expressed the view that the atrophy of the iris is a result of the mechanical stretching of this tissue, which causes a narrowing and occlusion of the radial arteries, thus producing a nutritional disturbance of this tissue in all portions other than that supplied by the lesser circle. He does not feel that the blocked angle could entirely explain the glaucoma. According to him, it is more likely that the elevated tension is caused by the loss of iris tissue and capillaries, for he regards these vessels as an important outlet for the aqueous.

Larsson has regarded the atrophy of the iris as a developmental anomaly, the starting point being a corectopia; the resultant secondary glaucoma finally causes tears in the iris tissue.

A local abiotrophy was assumed by de Schweinitz to be the cause of the iris atrophy; he also mentioned "premature senility" with death of cells.

Von Grosz agrees somewhat with de Schweinitz when he considers the condition as due to a hereditary feebleness of the iris, of a neurogenic character. As was to be expected, tuberculosis and syphilis have been offered as causes, the former being suggested by Lane, and the latter disease by the cases of Licskó and De la Vega.[24] This last author observed hole formation in the iris subsequent to a luetic chorioretinitis, and he believed it to be due to syphilitic changes of the uveal vessels. The view expressed by Hess, Harms, and Wood that it is a result of a chronic iridocyclitis is not shared by any of the later authors. However, it is well known that iris atrophy and defects can arise as a result of iritis or iridocyclitis.

This occurred in three cases reported by Franck[25] in severe iridocyclitis with posterior synechiae; as a result of the subsequent thinning, the atrophic iris tissue was split. In Franck's opinion this was a consequence of the mechanical factors operating on the atrophic iris tissue.

Conclusions

It is obvious in viewing these theories that there is no definite basis for the majority of them. As to the causes of the initial iris atrophy, virtually nothing is elucidated from either the microscopic or the clinical reports cited. The theories are purely speculative and obviously do not explain the condition. Concerning the glaucoma, the blocking of the chamber angle by the dense anterior synechiae easily explains this complication. I do not see the necessity for assuming that the disappearance of the capillary bed of the iris plays a role in the increased intraocular tension, for many cases have been seen clinically in which large areas of the iris were removed as a result of trauma or operative procedures, without a resultant rise of pressure.

1610 Eye Street, N.W.

Bibliography

[1] Johnson, G. L. Ophth. Rev., 1886, v. 5, p. 57.
[2] Hess, C. Klin. M. f. Augenh., 1892, v. 30, p. 103.
[3] Harms, C. Klin. M. f. Augenh., 1903, v. 41, p. 522.
[4] Wood, C. A. Ophthalmoscope, 1910, v. 8, p. 858.
[5] Lane, L. Ophth. Rec., 1917, v. 26, p. 285.
[6] Feingold, M. Amer. Jour. Ophth., 1918, v. 1, p. 1.
[7] Larsson, S. W. Klin. M. f. Augenh., 1920, v. 64, p. 510.
[8] Licskó, A. Klin. M. f. Augenh., 1923, v. 71, p. 456.
[9] Arnold, G. Klin. M. f. Augenh., 1923, v. 71, p. 723.
[10] Rieth, H. Klin. M. f. Augenh., 1936, v. 96, p. 349.
[11] Rochat, G. F., and Mulder, W. Brit. Jour. Ophth., 1924, v. 8, p. 362.
[12] Gifford, S. Amer. Jour. Ophth., 1926, v. 9, p. 548.
[13] de Schweinitz, G. E. Trans. Amer. Ophth. Soc., 1926, v. 24, p. 122.
[14] Griscom, J. M. Amer. Jour. Ophth., 1927, v. 10, p. 647.
[15] Ellett, E. S. Trans. Amer. Ophth. Soc., 1928, v. 26, p. 306.
[16] Waite, J. H. Amer. Jour. Ophth., 1928, v. 11, p. 187.
[17] Jeancon, E. Amer. Jour. Ophth., 1933, v. 16, p. 813.
[18] Barr, A. S. Arch. of Ophth., 1934, v. 12, p. 567.
[19] von Grosz, S. Arch. f. Augenh., 1936, v. 110, p. 111.
[20] McKeown, H. S. Arch. of Ophth., 1937, v. 18, p. 347.
[21] Fine, M., and Barkan, H. Amer. Jour. Ophth., 1937, v. 20, p. 277.
[22] Bentzen, C. F., and Leber, T. Arch. of Ophth., 1895, v. 41 (pt. 3), p. 208.
[23] Kreiker, A. Klin. M. f. Augenh., 1928, v. 80, p. 492.
[24] De la Vega. Klin. M. f. Augenh., 1923, v. 71, p. 44.
[25] Franck, A. Arch. f. Augenh., 1903, v. 47, p. 198.

COMPARATIVE STUDY OF BENZEDRINE, PAREDRINE, AND COCAINE WITH HOMATROPINE AS CYCLOPLEGICS*

EDWARD B. WEINMAN, M.D., AND F. BRUCE FRALICK, M.D.
Ann Arbor, Michigan

This study was undertaken because of a considerable number of inquiries as to the value of Benzedrine sulfate and homatropine solutions in producing paralysis of accommodation for refractive purposes. Homatropine and cocaine solution, homatropine and cocaine tabloids, Benzedrine (amphetamine) and homatropine solution, and paredrine and homatropine solution were used as cycloplegics and the residual amplitude of accommodation was measured at intervals to determine the duration and magnitude of the cycloplegic effect.

Only persons between the ages of 15 and 40 years were included in this study since the subjective method was used in measuring the amplitude of accommodation under the various cycloplegics. Those under 15 were believed to be unreliable for the subjective method of measuring the amplitude of accommodation because of poor coöperation. The amplitude of accommodation was measured according to the method of Duane.[1] A +3.00 D. sphere was added to the distance correction to bring the focus to about 33 cm. from the eye. Each patient was instructed to fixate upon a Jaeger test type no. 1 and move it towards his eye until blurring was first noted, obtaining the punctum proximum; then move it away until blurring was first noted, obtaining the punctum remotum. These distances were measured in centimeters from the eye and each measurement divided into 100 cm. to obtain the readings in diopters. The difference between the punctum proximum and punctum remotum in diopters designates the range of amplitude of accommodation present. Each eye was measured separately, and the time interval between instillation of the cycloplegic and measurement of accommodation noted.

HOMATROPINE AND COCAINE SOLUTION

Figure 1 shows the results obtained in 100 cases in which our routine cycloplegic of 4-percent homatropine hydrobromide with 0.5-percent cocaine hydrochloride in boric-acid solution was used. One drop of this solution was placed in the lower cul-de-sac at 10 minute intervals for six instillations and the patient instructed to keep the lids closed for a few seconds after each instillation. The amplitude of accommodation after six instillations varied between 0.7 D. and 1.6 D. In 24 hours the range of accommodation had not increased to over 2½ D. A sufficient number of these cases was

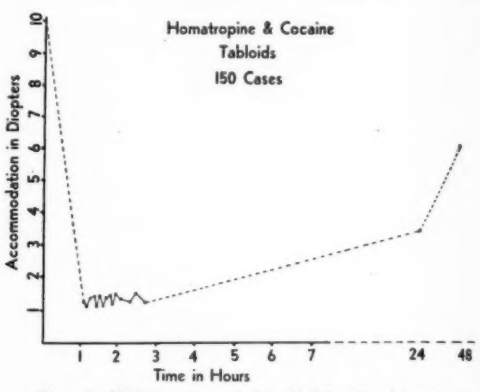


Fig. 1 (Weiman and Fralick). Residual accommodation in diopters, measured after six drops of a solution containing 4-percent homatropine hydrobromide and 0.5-percent cocaine hydrochloride in boric acid were instilled at 10-minute intervals in the cul-de-sac. Time measured from first instillation.

* From the Department of Ophthalmic Surgery, University of Michigan.

not carried over a period of days to determine when the accommodation returned to normal, but from our experience the symptoms last no longer than three days and postcycloplegic examinations were made at least four days after the use of this cycloplegic. An occasional case showed the accommodation still affected at this time.

The homatropine paralyzes the accommodation. Cocaine causes constriction of the dilator pupillae, but it also produces anesthesia of the superficial tissues of the eye. A serious objection to this form of cycloplegic is the occasional presence of corneal edema and staining, and the cornea frequently appears dull, making retinoscopy more difficult. However, good cycloplegia is obtained.

Homatropine and Cocaine tabloids

Similar results were obtained when homatropine and cocaine tabloids* were used. Figure 2 shows the results obtained in 150 cases followed in the same manner as when the solution of homatropine and cocaine was used. The residual amplitude of accommodation at the end of one hour varied between 0.9 D. and 1.6 D. The paralysis was greatest shortly after one hour following insertion of the tabloid but lasted several hours at less than 2 D. and did not return to normal for at least three days. These small wafers contain 0.02 gr. homatropine hydrochloride and 0.02 gr. cocaine hydrochloride, are about 0.5 mm. thick and 2 mm. in diameter and appear to go into solution almost immediately. One of these was inserted in the lower cul-de-sac and the patients were instructed to keep the lids closed for two minutes. All complained of foreign-body sensation and there was slight lacrimation and hyperemia. In some, 0.5-percent pontocaine was instilled before the tabloid was inserted but this was discontinued since it was of little benefit.

Corneal edema was not noted in any of this series but corneal anesthesia was present. The patients were allowed to keep the eyelids open after two minutes following instillation.

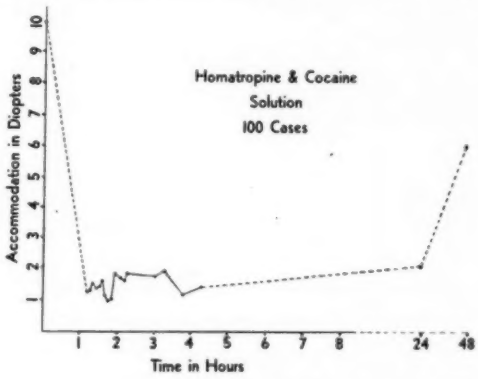


Fig. 2 (Weinmann and Fralick). Residual accommodation in diopters after insertion of a tabloid containing 0.02 gr. homatropine hydrochloride and 0.02 gr. cocaine hydrochloride in lower cul-de-sac.

Benzedrine and Homatropine solution

Since Myerson and Thau[2] published their work on Benzedrine in 1927, Beach and McAdams,[3] Powell and Hyde,[4] and Sudranski[5] have published their findings. In general they all agree that Benzedrine and homatropine solutions can be used together to produce cycloplegia for refractive purposes, and that it has the advantage of permitting quick recovery.

In this series in which 731 cases were examined under Benzedrine** and homatropine, we find similar results. Our technique differed from that used by the

* Furnished by courtesy of Burroughs Wellcome and Company, New York.

** Furnished by courtesy of the manufacturers, Smith, Kline, and French Company, Philadelphia. The preparations were their product—Benzedrine (amphetamine) and Benzedrine sulfate (amphetamine sulfate).

aforementioned authors. A single drop of a solution containing 5-percent homatropine hydrobromide and 1-percent Benzedrine sulfate in distilled water was used. The patients were instructed to tilt their heads back and look down, the eyelids were held away from the globe and a single drop was placed upon the limbus above and allowed to flow over the cornea. The eyelids were kept closed for one-half minute following this. No difference was noted in the cyclopeglic effect, whether the patients stayed in a dark room, or went into the sunlight, or if they kept their lids open or closed. Age was not an important factor, but children under 15 years of age were not included in this study. In a few cases 1-percent atropine sulphate was substituted for the homatropine hydrobromide in children under 15 years, but the amount of residual accommodation varied with the amount of coöperation.

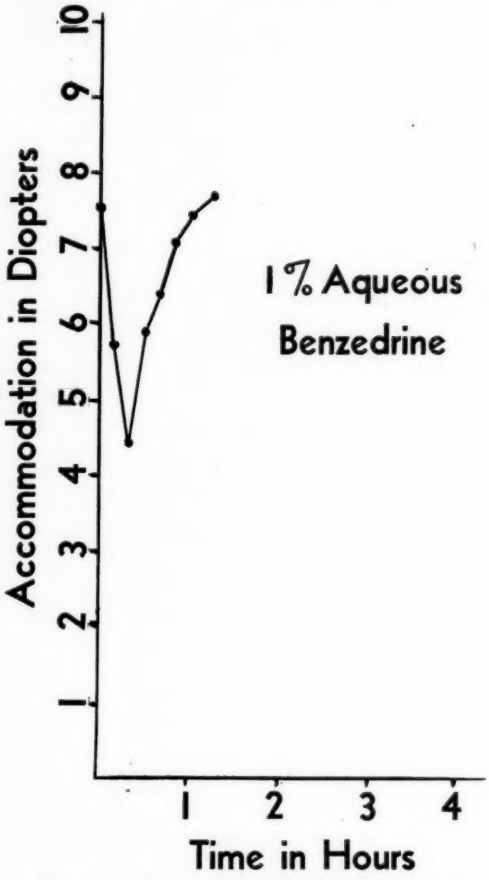


Fig. 3 (Weinmann and Fralick). Effect upon accommodation of one drop of a 1-percent aqueous-Benzedrine-sulfate solution instilled over the cornea.

According to Myerson and Thau,[6] Benzedrine sulfate in solutions ranging from 0.25 to 10.0 percent when instilled into the cul-de-sac of the eye widens the palpebral fissure, dilates the pupil, decreases accommodation, reduces sensitivity of the cornea, and constricts the vessels of the retina. Some workers believe that the accommodation is not disturbed when Benzedrine sulfate alone is used. We found, however, that in using one drop of a 1-percent aqueous solution of Benzedrine the accommodation was disturbed as shown in figure 3. The greatest effect was after 30 minutes, and there was rapid recovery. The effect upon accommodation seemed to precede the effect upon the pupil. Greatest pupillary dilatation was found at about 30 to 45 minutes. Full dilatation of the pupil was not uniformly obtained with this dilution of Benzedrine sulfate.

When Benzedrine sulfate was used with homatropine hydrobromide the maximum loss of accommodation was found at 55 minutes after instillation, and the recovery was almost complete after eight hours. Most people could resume reading seven hours after its use. A few still complained of some blurring on close work the following morning when the medication was given in the late afternoon, but none had complaints after 18 hours when only one drop was instilled.

Figure 4 shows the mean results in 731 cases in which one drop of the solution containing 1-percent Benzedrine sulfate and 5-percent homatropine hydrobromide was used. Several cases were followed at 15 minute intervals during the early stages of the cycloplegia,

some at one-half-hour intervals, in most there were only one or two determinations. Within 16 to 21 minutes after instillation blurring of vision was noted. The pupils were sluggish after 15 minutes, well dilated but active at one-half hour, widely dilated and inactive at 45 minutes, and in general no reaction could be noted until four hours after instillation. The corneas remained clear and bright and the pupils were more widely dilated than when homatropine and cocaine were used. No allergic reactions were noted. An occasional person complained of a slight burning at the time of instillation which wore off within one minute. None complained of nasal irritation. Intraocular tension was not taken

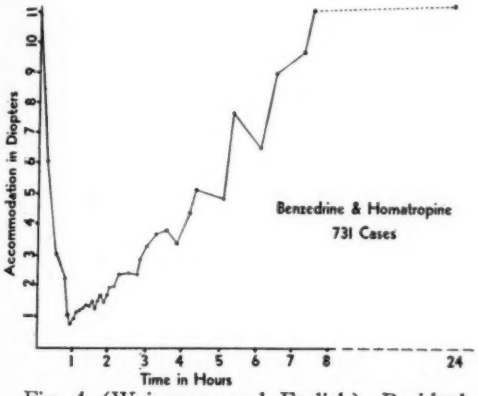


Fig. 4 (Weinmann and Fralick). Residual accommodation in diopters after instillation of one drop of 1-percent Benzedrine-sulfate and 5-percent homatropine-hydrobromide solution upon the cornea. The average remaining accommodation at intervals was plotted to show the time of maximum cycloplegia and the gradual return of accommodation.

tonometrically but no known pathologic increase was found. All patients agreed that this was more desirable than the routine cycloplegic.

Investigation was made to determine any variation in amount of amplitude of accommodation according to color, age, sex, type of error, and wearing of correction after the use of Benzedrine-homatropine cycloplegia. Brunet, or darkly pigmented, individuals did not obtain so great a paralysis of accommodation as did the more blond individuals. One drop was not sufficient in Negroes, from two to six instillations being required. Negroes were not followed to determine the period of recovery, but at time of refraction the amplitude of accommodation was measured to make certain that adequate cyclo-

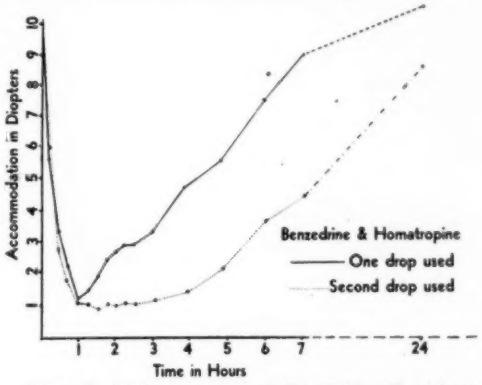


Fig. 5 (Weinmann and Fralick). Residual accommodation in diopters measured after instillation of one drop of a 1-percent Benzedrine-sulfate and 5-percent homatropine-hydrobromide solution, shown by the solid-line curve, as compared with the effect upon accommodation when a second drop was given at the end of one hour, as shown by the dotted-line curve.

plegia was obtained. Age, sex, error of refraction, and wearing of correction showed no uniform variation.

When more than one drop of the Benzedrine-and-homatropine solution was instilled during the interval before refraction, certain variations of amplitude of accommodation were found. This was probably due to the cumulative effect of the homatropine. The return of accommodation was slower. Figure 5 shows the effect of a second drop given at the end of one hour as compared with the use of one drop alone. The combined action of these two drops given at a one-hour interval produced adequate cycloplegia for refractive purposes for about three hours after the second drop. We now employ this method of giving a second instillation if we are not able to refract the

individual between 50 and 90 minutes.

Figure 6 shows the necessity of placing the drop of solution over the cornea and may explain some of the variable results obtained by some users. In a few of our first cases we instilled one drop of the solution into the cul-de-sac and found

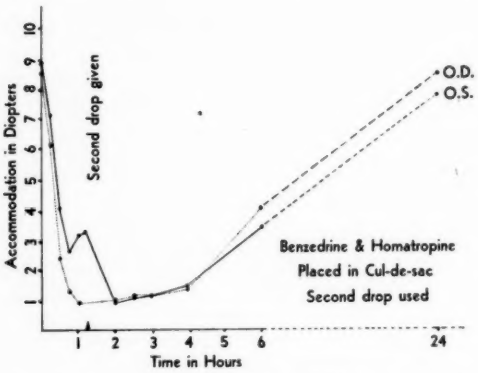


Fig. 6 (Weinmann and Fralick). Residual accommodation in diopters measured after instilling one drop of 1-percent Benzedrine-sulfate and 5-percent homatropine-hydrobromide solution in the cul-de-sac, showing the poor cycloplegia obtained in the left eye, the drop having been given in the right cul-de-sac first. Apparently an insufficient amount of the solution came into contact with the cornea. A second instillation was made over the cornea after 75 minutes, and good cycloplegia was obtained.

the amplitude of accommodation to be variable. It was noted that better cycloplegia was obtained in the eye in which the cycloplegic was instilled first. As shown in figure 6, excellent cycloplegia was obtained in the eye in which the solution was first instilled, the right eye, but apparently not enough of the medication was absorbed in the left, and a second drop was instilled over the cornea of both eyes.

Paredrine and Homatropine solution

Paredrine,* a sympathomimetic drug closely related to Benzedrine, was suggested by its manufacturer as a substitute for Benzedrine as a synergist with homatropine for cyclopegic effect: According to Abbott and Henry,[7] paredrine acts as a mydriatic, the maximum effect being noted in 60 minutes and lasting about six hours; does not cause an increase in intraocular tension; is nonirritating to the mucous membranes; does not cause loss of accommodation; and the mydriasis could be neutralized with miotic drugs. Tassman[8] instilled one drop of 4-percent homatropine hydrobromide and three minutes later one drop of 1-percent aqueous paredrine hydrobromide in patients over 16 years of age; one drop of atropine sulphate followed in three minutes by one drop of 1-percent paredrine hydrobromide if under 16; and a second drop of paredrine was instilled when desired. He found no increase in the intraocular tension and maximum dilatation of the pupil in 40 to 50 minutes with return to normal in four

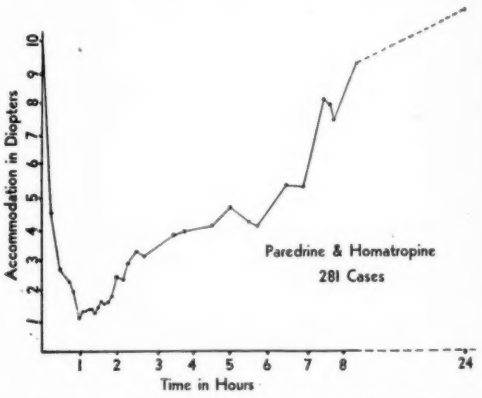


Fig. 7 (Weinmann and Fralick). Residual accommodation in diopters measured after instilling one drop of 1-percent paredrine-hydrobromide and 5-percent homatropine-hydrobromide solution over the cornea. Maximum cycloplegia was obtained at 60 minutes and accommodation returned to normal after eight hours.

to five hours. The residual accommodation in his cases was well under one diopter.

In our study with paredrine we found similar results but we did not use repeated instillations. A solution of 1-per-

* Supplied by Smith, Kline, and French Company, Philadelphia, for this study.

cent paredrine hydrobromide and 5-percent homatropine hydrobromide in boric acid was used in the same manner as was the Benzedrine and homatropine solution described in the preceding section. The results in 281 cases as shown in figure 7, were very similar. The greatest cycloplegic effect was obtained at 60 minutes and recovery was not quite so rapid as when the Benzedrine was used. Accommodation returned to about normal in 8½ hours. No one had complaints after 18 hours.

In no case was a pathologic increase in the intraocular tension noted, nor any allergic symptoms. The pupils were fixed after 45 minutes and began to show activity after five hours. Most of the patients could read after eight hours. As with the Benzedrine and homatropine solution, the corneas remained clear and bright, pupils were markedly dilated, and adequate cyclopegia for refraction was obtained between 60 and 90 minutes after instillation. Adequate cycloplegia could not be obtained in Negroes without repeated instillations, and blonds were more susceptible than brunets. No considerable variations in cycloplegic effect was noted in the various age groups over 15 years.

Figure 8 shows the results of a second instillation of this solution as compared with those when only one drop was used. The second instillation was given 15 minutes after the first. The cycloplegia obtained did not seem to be any greater, but when two drops were used it was more lasting, giving a longer period of safety in which to carry out the refraction. In the cases in which two instillations were given there was complete return of accommodation after 24 hours.

Conclusions

Four-percent homatropine hydrobromide and 0.5-percent cocaine hydrochloride solution given at 10-minute intervals for six instillations give a residual accommodation ranging between 0.7 and 1.6 D. which lasts for several hours and allows a long period in which to complete the refraction. The solution, in many

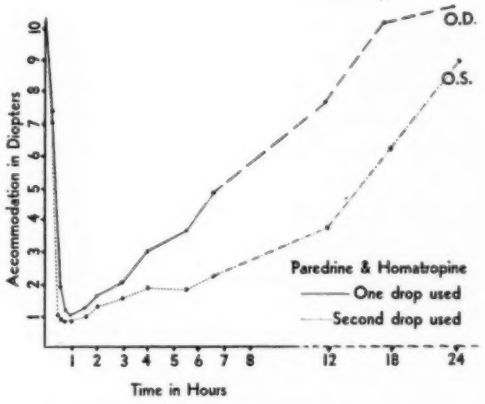


Fig. 8 (Weinmann and Fralick). Accommodation was measured at intervals after instillation of one drop of a 1-percent paredrine-hydrobromide and 5-percent homatropine-hydrobromide solution over the corneas of each eye; a second drop was placed over the cornea of the left eye 15 minutes later. The cycloplegia obtained from the two drops is more lasting than if only one drop is used but the accommodation has approximately returned to normal after 24 hours.

cases, causes the cornea to be dull, making retinoscopy more difficult, and occasionally the corneal epithelium will retain fluorescein stain, showing evidence of drying from its use.

One-fifthieth grain homatropine hydrochloride and 0.02 grain cocaine hydrochloride tabloids give approximately the same paralysis of accommodation as obtained by the repeated instillations of the solution; only one insertion is necessary. There is an adequate depth and period of cycloplegia to carry out the refraction. With the use of the tabloid there is a foreign-body sensation which cannot be completely relieved with topical pontocaine anesthesia. The necessity of only one insertion allows the patient to be excused for at least an hour before returning for refraction.

One-percent Benzedrine sulfate and 5-percent homatropine hydrobromide can be combined in one solution, and one drop, if placed over the cornea, will give cycloplegia as great as that obtained from the homatropine and cocaine solution or tabloid, but the cycloplegia is not lasting. Optimum time for refraction after the instillation of the benzedrine and homatropine solution is 55 minutes; most of the cycloplegic effect has worn off after 7 hours with return to normal after 18 hours. The pupil is more widely dilated with this solution than with the homatropine and cocaine mixtures and the cornea remains clear and bright after its use. Benzedrine sulfate, 1-percent aqueous solution, does have a slight cycloplegic effect.

One-percent paredrine hydrobromide with 5-percent homatropine hydrobromide acts similarly to Benzedrine and homatropine and is said to be more satisfactory, as it does not cause an increase in the intraocular pressure. The action of paredrine and homatropine upon accommodation is slightly more prolonged than that of Benzedrine and homatropine, but patients who received one drop of the solution may resume their occupation after 18 hours without complaints. Paredrine is slightly less irritating when used on the conjunctiva than is Benzedrine.

Two instillations of Benzedrine and homatropine solution or paredrine and homatropine should be given if there is any question of whether the initial drop flowed over the cornea or if it is impossible to test the refraction of the patient between 60 and 90 minutes after the initial instillation.

Quick recovery from cycloplegia is an important factor to many patients and can be obtained with either the Benzedrine and homatropine or paredrine and homatropine solutions. Either of these solutions gives greater cycloplegia in blonds than in brunets, but if used in Negroes, repeated instillations are necessary.

Postcycloplegia examinations can be made on the following day if Benzedrine and homatropine or paredrine and homatropine solutions are used, but it is necessary to wait until the fourth day if homatropine and cocaine tabloids or solution is used.

University Hospital.

References

[1] Duane, A. Fuchs's Textbook of ophthalmology. Ed. 5, New York, J. B. Lippincott Co., 1917, p. 857.

[2] Myerson, A., and Thau, W. Human autonomic pharmocology: the effect of cholinergic and adrenergic drugs on the eye. Arch. of Ophth., 1937, v. 18, July, p. 78.

[3] Beach, S. J., and McAdams, W. R. Benzedrine in refraction: Preliminary report. Trans. Amer. Ophth. Soc., 1937, v. 35, pp. 221-226.

———. Benzedrine in cycloplegia: Further report. Amer. Jour. Ophth., 1938, v. 21, Feb., pp. 121-124.

[4] Powell, L. S., and Hyde, M. E. Effect of benzedrine sulphate solution on cycloplegia. Preliminary report. Jour. Kansas Med. Soc., 1938, v. 39, Jan., pp. 1-4.

———. Action of benzedrine sulphate solution. Jour. Kansas Med. Soc., 1938, v. 39, Oct., pp. 434-435.

[5] Sudranski, H. F. Evaluation of homatropine-benzedrine cycloplegia. Arch. of Ophth., 1938, v. 20, Oct., pp. 585-596.

[6] Myerson, A., and Thau, W. Human autonomic pharmocology: Effect of benzedrine sulphate on Argyll Robertson pupil. Arch. Neurol. and Psychiat., 1938, v. 39, April, pp. 780-788.

[7] Abbott, W. O., and Henry, C. M. Paredrine; a clinical investigation of a sympathomimetic drug. Am. Jour. Med. Sci., 1937, v. 193, May, pp. 661-673.

[8] Tassman, I. S. Paredrine in cycloplegia. Amer. Jour. Ophth., 1938, v. 21, Sept., pp. 1019-1024.

BILATERAL PLEXIFORM NEUROMATA OF THE CONJUNCTIVA AND MEDULLATED CORNEAL NERVES

Report of a case*

Martin P. Koke, M.D., and Alson E. Braley, M.D.
Iowa City, Iowa

Neurogenic tumors of the eye and its appendages are frequently referred to in the literature, most of the reports being concerned with extensive neurofibromatosis in which elephantiasis of the lids, proptosis, and wide involvement of the surrounding structures are often described. With neurogenic ocular tumors there is commonly associated a generalized neurofibromatosis of von Recklinghausen, and occasionally corneal-nerve changes have been noted. Snell[1] (1903) reported three cases of plexiform neuroma involving the lids and eye. In a report of the pathology of these cases, Collins described one case with groups of elongated cells in the substantia propria of the cornea; he was of the opinion that these continuous tracks of elongated cells were due to enlargement of the corneal nerves. Collins and Batten[2] (1905) found similar cell structures in the cornea associated with neurofibromatosis of the eyeball and its appendages and again expressed the view that these linear tracks of elongated cells were thickened nerve fibers. Sutherland and Mayou[3] (1907), reporting a case of neurofibromatosis of the fifth nerve, observed a number of white streaks in the periphery of the cornea which they thought were enlarged ciliary nerves. Groups of elongated cells beneath the anterior limiting membrane in a similar case were described as thickened nerves by Hine and Wyatt[4] (1928).

Guist[5] (1920) reported small neurofibromata in the bulbar and palpebral conjunctiva in von Recklinghausen's disease. He also quoted von Michel, who observed a similar tumor on the bulbar conjunctiva in a case of neurofibroma of the upper lid. The literature on neurogenic conjunctival tumors, like that on enlarged corneal nerves, is scant.

The case presented here is unique in that no neurogenic tumors were found except the small bilateral conjunctival nodules and a pheochromocytoma of the right adrenal gland.

Case history

A 28-year-old white male was admitted to the Medical Department** in September, 1938, and found to have paroxysmal hypertension. In the attacks of hypertension, the blood pressure often reached 260/140 and remained elevated several hours. After a tentative diagnosis of adrenal tumor was made, the patient was transferred to the Surgery Department and at operation, an egg-sized tumor involving the right adrenal gland was removed. This proved to be a pheochromocytoma. Since operation, the patient has had no hypertensive attacks, the blood pressure having been 115/75 or thereabout for many weeks.

Ocular history. During an attack of measles, 20 years previously, the patient's eyes were swollen shut for three days, but he apparently made a complete recovery. In the spring of 1934, while working (disking) in dense clouds of dust, his eyes smarted and became inflamed. A diagnosis of granulated eyelids was made, and the local physician excised approximately 25 small tumors from the palpebral conjunctiva. The

* From the Department of Ophthalmology, College of Medicine, State University of Iowa.

** Full report of this case will be published by the Department of Medicine.

patient had been unaware of the lid tumors. No further eye trouble was experienced except that each following spring the patient's eyes were very sensitive to cool winds; however, this irritation was relieved by tight goggles.

Ocular examination. On the palpebral conjunctiva of each lid of both eyes, four or five small yellowish-gray tumors were found; these were near the lid border and in some instances extended to the intermarginal space. They were 1 to 2 mm. in diameter with an elevation of approximately 0.5 mm. Most of them were discrete but some had coalesced. Many of the tumors were sessile with a smooth surface; however, a few showed elevated ridges that gave the impression of an underlying coiled mass. In the bulbar conjunctiva were three or four tumors located approximately 1 mm. from the limbus, in the area of the palpebral fissure. They were similar to those in the lids except for a greater depth of color. The bulbar tumors simulated pinguecula but histologically were neurogenic tumors. The conjunctiva was normal otherwise.

The corneae were of normal dimensions and gross contour. The superficial layers showed some vascularization at the periphery; the vessels were entirely superficial and coming from the conjunctival circulation extended onto the corneae for a distance of approximately 1.5 mm. above and 0.5 mm. below. Biomicroscopic examination of the corneae under low power and with a broad beam of light revealed a network of fine grayish-white anastomising and interlacing fibers situated in the stroma. Several of the main trunks, which were 5 or 6 mm. in length, could be seen without magnification. In the periphery these whitish-gray structures were situated at the junction of the anterior two thirds and the posterior one third of the corneal lamellae, but as they extended toward the pupillary area they diminished in caliber and became more superficial. There were two sets of the whitish-gray structures both of which branched dichotomously. The larger set sent branches into the pupillary area, while the smaller more superficial set branched in the periphery. Some of the branches arched backward toward the limbus and anastomosed with similar branches while others extended into the more superficial stroma and disappeared. At a few of the nerve forks there was a slight bulbar enlargement.

Corneal microscopy with the high power and narrow slit showed the individual structures of the rete to be round or oval, the latter being flattened anteroposteriorly. They were composed of a thick white wall surrounding a fine, moderately clear fibril. The more minute of these structures, particularly those forming the superficial plexus and the end branches of the larger fibers, did not have the white covering. It was not uncommon to see branches coming directly forward from some of the main trunks and disappearing in the superficial corneal stroma. The caliber of the larger fibers was somewhat irregular, due to thickenings of the sheath, but no definite nodules were seen. In every instance their diameter diminished as they passed from the periphery toward the pupillary area.

The blood vessels of the pannus were very superficial, arising from the conjunctival circulation and extending onto the cornea just beneath the epithelium, and even after instillation of 5- and 10-percent dionin, they remained entirely unrelated to the deeper white fibers. In no instance did blood enter the clear centers of the large white trunks.

Fundoscopic examination was essentially normal, and no medullated retinal nerve fibers were found. The visual fields were normal. The corrected vision, O.U. was 6/6.

Pathology. Four tumors from the conjunctiva, one from the bulbar and three from the palpebral area, were examined histologically. They were sessile, round or oval, and approximately the same size. The three removed from the palpebral conjunctiva were covered by a thin layer of stratified epithelium in which shallow glands and a few goblet cells were seen. In the substantia propria, immediately beneath the epithelium, were a moderate

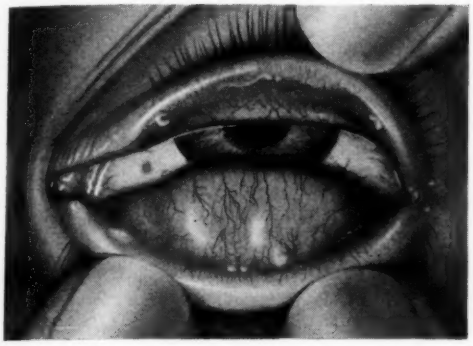


Fig. 1 (Koke and Braley). Conjunctival tumors.

number of capillaries. Situated in the subconjunctiva was an oval nodule composed of bundles of elongated spindle-shaped cells cut in cross section, obliquely, and longitudinally; their nuclei were long, slender, deeply stained structures. The cells were surrounded by pale-staining structures and, on seeing a bundle cut in cross section, it gave one the impression of a nerve surrounded by large amounts of perineurium. A few ganglion cells were found scattered between and incorporated within the nerve bundles; they were large, pale-staining cells with vesicular nuclei containing a prominent nucleolus, and cytoplasmic granules closely simulating Nissl bodies. In Held stain the tumor showed many medullated nerve fibers, the sheaths of which varied in thickness, some being several times as thick as normal. In Mallory's stain the entire tumor stained red grossly, while the subconjunctiva and tunica propria took the blue stain. From this one would suspect that the tumor was largely of epithelial origin; however, scattered through the tumor were fine blue connective-tissue strands much like epineurium. The tumor mass appeared to be made up of a coiled nerve. In silver stain it was composed almost entirely of axis-cylinders. In many areas the axis-cylinders formed interlac-

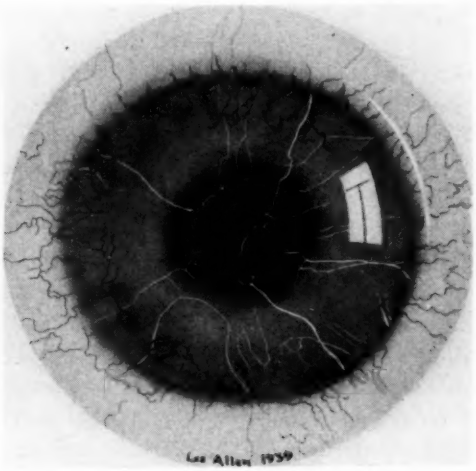


Fig. 2 (Koke and Braley). Large corneal nerves.

ing bundles, but in some they had no definite arrangement. The tumor removed from the bulbar conjunctiva near the limbus was smaller but showed a similar type of histology.

Comment

These conjunctival tumors, being composed of nerve fibers and a few ganglion cells, are neuromata and, since they consist chiefly of hyperplastic nerve fibers and connective tissue, should be classified as plexiform neuromata. Myelinic or amyelinic neuroma, the names used by Ewing[6] for overgrowths of medullated or nonmedullated nerve fibers, contain less connective tissue, while neurofibromata have fewer nerve fibers than the tumors

here reported. The nerve fibers in neurofibromata are passive and gradually atrophy so that, unlike plexiform neuromata, their bulk is connective tissue. The origin of neuromata according to Bruce and Dawson,[7] who disproved the theory that medullated nerve fibers cannot develop without the influence of ganglion cells, is widely scattered cells of embryonic residue. They found that these cells and the resulting nerve fibers had a tendency to develop in the adventitia of blood vessels. Their studies, however, were made of neuromata from the medulla, cord, and pia. The tumors described here are undoubtedly derived from the nerve endings of the fifth nerve and are thus closely related to the changes in the corneal nerves. Since the axis-cylinders in the conjunctival tumors showed proliferation of the neurolemma and myelinization, it is justifiable to consider the corneal nerve changes as due to a similar process.

We are of the opinion that the gray-white structures in the cornea are medullated corneal nerves and are part of the disturbance manifested by the neurogenic conjunctival tumors of the fifth nerve.

Fischer[8] and others who have described medullated retinal nerve fibers in several cases of von Recklinghausen's disease believe that this association is too frequent to be mere coincidence. Rosenthal and Willis,[9] considering the association of pheochromocytoma and neurofibromatosis, found five such cases in the literature and reported another; they too felt that the frequency of this association was too great to be fortuitous. No ocular abnormalities were reported in their cases. If there is a relationship between the chromaffin cells of the adrenal and neurogenic tumors, perhaps the abnormalities of the corneal nerves and conjunctival tumors are a response to the products of the adrenal tumor.

Conclusions

In a patient with paroxysmal hypertension that was due to a pheochromocytoma of the adrenal gland, bilateral plexiform neuromata were found in the palpebral and bulbar conjunctivae, and there were bilateral medullated corneal nerves.

Bibliography

[1] Snell. Trans. Ophth. Soc. U. Kingdom, 1903, v. 23, p. 157.
[2] Collins and Batten. Trans. Ophth. Soc. U. Kingdom, 1905, v. 25, p. 248.
[3] Sutherland and Mayou. Trans. Ophth. Soc. U. Kingdom, 1907, v. 27, p. 179.
[4] Hine and Wyatt. Brit. Jour. Ophth., 1928, v. 12, p. 513.
[5] Guist. Klin. M. f. Augenh., 1920, v. 65, pt. 2, p. 850.
[6] Ewing. Neoplastic diseases. Philadelphia and London, W. B. Saunders Co., 1928, ch. 23.
[7] Bruce and Dawson. Rev. Neur. and Psych., 1913, v. 11, p. 235.
[8] Fischer. Dermat. Zeit., 1924-1925, v. 42, p. 143.
[9] Rosenthal and Willis. Jour. Path. and Bact., 1936, v. 42, p. 599.

MIDLINE NOTCHING IN THE NORMAL FIELD OF VISION*

HUGO L. BAIR, M.D., AND ROBISON D. HARLEY, M.D.
Rochester, Minnesota

In otherwise normal fields one frequently finds a small midline contraction in the upper field. Occasionally this is not directly in the midline but may be slightly to one side or the other. The presence of such contractions raises the questions: Are the fields normal? What interpretation is to be given to the contractions?

In the literature of perimetry, reference has frequently been made to contractions in the limits of the upper field. These have referred to altitudinal contractions affecting both halves of the field. They have not included the type of superior midline notching to which we refer. The only report in which this finding is described is by Lanchester,[1] who also was unable to find any previous reference to it. Lanchester described this as a rift or hiatus which could be found extending vertically above or below the fixation point. He demonstrated it by having the subject fixate a given point and then note the disappearance of several points lying in a vertical line directly above or below it. Points placed in the lateral field remained in view constantly while the vertically placed points disappeared. He did not consider the possibility that the disappearance of the vertically placed points was an example of Troxler's phenomenon. His account did not bring out any relationship of such a vertical hiatus to clinical perimetry. He attributed the presence of the hiatus to insufficient overlapping of the two halves of the visual field, and he explained the failure of the two halves to overlap sufficiently on the basis that each half was isolated from the other half in its cortical representation.

We have found a superior midline notching of the visual fields so frequently in otherwise normal fields that we feel it is desirable to call attention to its occurrence and possible interpretation. It has been found mostly in examination of the limits of the field on the one-meter tangent screen. Occasionally, it has been found in examination with the perimeter of 33 cm. radius. Rarely have we found an exactely similar defect in the midline below.

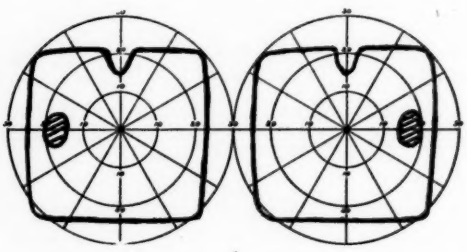
In order to demonstrate the occurrence of this notching we investigated a series of seven young normal adults. In each case the vision was 6/5. Two subjects wore glasses. The ocular fundi were entirely normal and vascular anomalies that could cause an angioscotoma in the midline were not present. The visual fields of these subjects were normal in all other particulars. Both eyes in each of the seven subjects were examined and superior altitudinal notching was found in the field of each eye in every case. Examination was made on the one-meter black-felt tangent screen illuminated by the Bausch and Lomb tangent-screen illuminator. A white bead, 1 mm. in diameter, mounted on a stick coated with black felt velure was used as a test object. Against the black-felt screen the holder for the bead was indistinguishable from the background. In order to prevent any inequalities of illumination of the bead in different parts of the field, the holder was held and moved vertically with the bead at its lower end. A white bead 3 mm. in diameter was employed for fixation. The subject's upper lids were elevated by the

* From the Section on Ophthalmology, The Mayo Clinic.

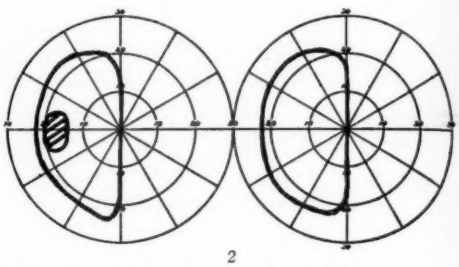
examiner's thumb when there was any question whatever of the lids or lashes causing any obstruction of the upper field. Such an obstruction by the lashes, the lids, or the brow, however, would not cause an isolated midline contraction but would cause a more uniform altitudinal contraction across the entire upper field.

A typical example that illustrates the nature and extent of these notchings is shown in figure 1. The apparently squared shapes of the fields represent, of course, not the true shapes, but the shapes imposed by the limits of the square area of illumination on the tangent screen. In two of the subjects the fields were also examined with the illumination of the tangent screen and the test object reduced to 3 percent of its usual value. Under this condition the notching was also demonstrated, even though the total extent of the field was smaller. The factors important in demonstrating this superior midline notching seem to be excellent maintenance of fixation, coöperation and intelligence of the subject, approximately normal visual acuity, and delicacy in the technique of the examination.

Inasmuch as this phenomenon appears to be part of the normal field, its explanation must be found in some normal anatomic or physiologic disposition of the visual elements corresponding to its position in the field. An obvious explanation would be that the midline of the retina below the fovea was not so densely populated with rods and cones as the parasagittal retina, owing to the presence of the inferior choroidal fissure during fetal life. Against this explanation, however, is the fact that the inferior fissure extends downward from the disc and not from the macula. A defect caused by it would be expected to extend upward from the blind spot instead of being above the fixation area. That the defect does not represent an angioscotoma is proved by the exclusion of any vessels in the retina whose position and size might cause it.



1



2

Fig. 1 (Bair and Harley). Typical superior midline notching in normal visual fields.
Fig. 2 (Bair and Harley). Complete right homonymous hemianopia, showing the shape of the remaining "unit fields."

Troxler's phenomenon is excluded as a cause by the consistent appearance and constant position of the defect. In the method by which Lanchester demonstrated the defect, the occurrence of Troxler's phenomenon was not excluded, however.

Lanchester's hypothesis that the defect represented a failure of the right and left visual fields to overlap would appear to explain it, but we would carry this explanation further. We consider that each half of the visual field constitutes an anatomic unit corresponding to the anatomic unity of its cortical representation. The representation of each unit field is to be found in the visual cortex of the

opposite occipital lobe. It is then reasonable to suppose that in the retinal counterpart of each unit half-field there is a decreasing density of retinal elements at all marginal points, including all of the medial or midline margin as well as the periphery. In the macular area, however, the elements are so dense that a decrease in their density comparable to the proportional decrease in density in the peripheral retina is so small that its effect on visual sensibility is imperceptible and cannot be demonstrated.

Such unit fields are seen in the half-fields remaining in cases of complete homonymous hemianopia that are the result of postchiasmal interruptions of one visual pathway (fig. 2). In these there is practically always found a gradually increasing departure of the medial limits of the remaining field from the midline above and below and fixation point. Putting together two such fields remaining on opposite sides so that the fixation points come together would give essentially the picture of the midline notching.

Recent exact counts of the density of rods and cones in the retina have been made by Østerberg.[2] The greatest density of cones was observed at the macula. Peripherally the cones were decreased but the diminution was less in the upper nasal than in the lower temporal part of the retina. At a distance of 5 to 6 mm. from the macula was found a pronounced ring-shaped zone of maximal rod density. Peripheral to this zone the rod count fell off, but in such a manner that all of the upper and nasal part of the retina was more populated than the lower and temporal part. No actual counts were made in the area corresponding to the midline of the field. This work, however, suggests a slight relative shortage of retinal elements in the area corresponding generally to the superior field, which would explain the greater demonstrability of the midline notching above than below.

Conclusions

Normally a midline notching may be found in the limits of the visual field. Its demonstration requires a coöperative, intelligent subject, steady fixation, and delicate technique. Its explanation, we believe, lies in a thinning of percipient elements in the part of the retina corresponding to the medial border of each half-field. This postulated thinning of percipient elements is based on the conception of each half-field as a unit field because of its separate cortical representation. Being a unit field, its retinal counterpart would have a thinning of percipient elements along all of its borders. Along the macular border the thinning is so slight in proportion to the normal density of rods and cones that its effect is inappreciable.

References

[1] Lanchester, F. W. Discontinuities in the normal field of vision. Jour. Anat., 1934, v. 68, Jan. pp. 224-238.

[2] Østerberg, G. Topography of the layer of rods and cones in the human retina. Acta Ophth., 1935, v. 13, Suppl. 6, pp. 29-102.

NOTES, CASES, INSTRUMENTS

BENIGN CYST OF THE OPTIC DISC

HENRY L. HILGARTNER, JR., M.D., F.A.C.S.
Austin, Texas

A case of benign cyst of the optic nerve is presented, which has been under observation since June 7, 1934, at which time the fundus was photographed with panchromatic films. The condition has not changed since the original photograph in black and white was taken.

Case report. J. E., a white boy aged 12 years, was first examined on June 7, 1934. The members of the family noticed that the vision in the right eye was not so good as that in the left. The patient was seen in consultation by Dr. H. L. Hilgartner, Sr., and Dr. Alvin Dinwiddie. The eyes were normally prominent; the pupils reacted to light; the cornea, sclera, and iris in each eye were normal. Vision in the right eye was ability to detect hand motions at 18 inches; left eye, 20/16. The right eye turned outward 15 degrees.

Through the dilated pupil, the fundus of the right eye appeared to be clear. The disc had a white mass projecting from the center, the tip of which was clearly seen with a plus 5 D. lens. The mass was about one third the size of the disc. No blood vessels could be seen associated with the mass. Extending from the 7-o'clock position there were distinct and discrete pigmented areas that made an arc surrounding the disc—much denser in the macular region. Scattered throughout the equator could be seen very small and discrete pigmented areas, particularly in the lower quadrant. There seemed to be some edema between the two upper temporal vessels. The blood vessels were somewhat smaller in size than in the other eye. The left fundus was normal.

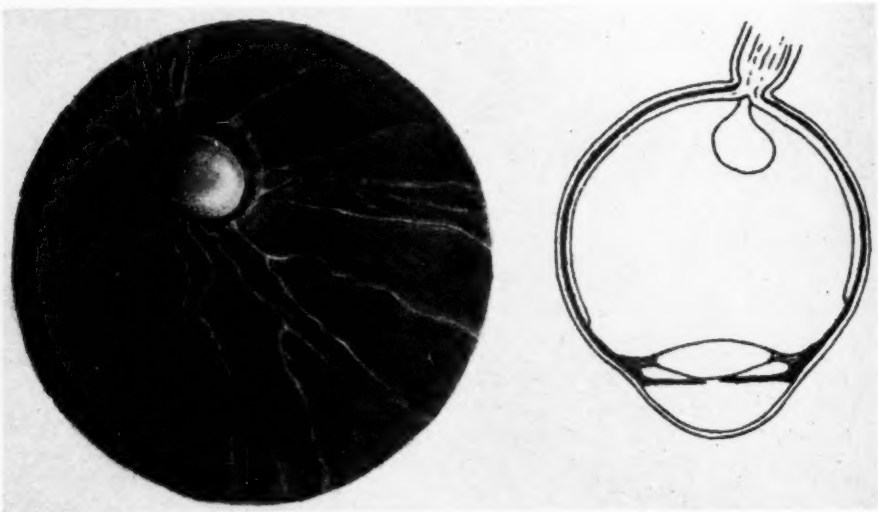


Fig. 1 (Hilgartner). Drawing and schematic drawing of cyst of optic disc.

On February 7, 1939, the patient returned at my request and examination of the right eye showed that apparently no change had taken place since 1934.

Comment. In the literature, only several reports are available in which the cyst resembled that of the case in ques-

tion. There are numerous references in which the basic condition was a sarcoma, a glioma, or a neurofibroma.

In 1863, A. Rothmund[1] reported a case of cystic degeneration of the optic nerve in which the tumor protruded from the left ocular cavity. Microscopic examination revealed numerous myxomatous cysts of various sizes, separated by bands of fibrinous tissue.

The case that Bane[2] reported in 1918 was that of a cyst of the dural sheath of the optic nerve, retrobulbar in position.

All of the cases that Verhoeff[3] reported in 1921-22 differ from my case in regard to the position and appearance of the tumor mass. He was also able to give the histological findings in 11 of the cases. At the present time there is no reason to enucleate the eye of my patient.

Levine, in 1932, reported a case[4] of acquired cyst of the optic disc which resembles this case. He quoted from Durst in describing the difference between a congenital and an acquired cyst. The main points are that the congenital anomaly is associated with other congenital changes, and the blood vessels are empty strands. The acquired cyst shows evidence of recent or old inflammation.

REFERENCES

[1] Rothmund, A. J. Neurom (cystose Degeneration) des Sehnerven. Klin. M. f. Augenh., 1863, v. 1, pp. 261-264.

[2] Bane, W. C. Cyst of dural sheath of optic nerve. Amer. Jour. Ophth., 1918, v. 1, pp. 17-20.

[3] Verhoeff, F. H. Primary intraneural tumors (gliomas) of the optic nerve. A histologic study of 11 cases, including a case showing cystic involvement of the optic disc, with demonstration of the origin of cytoid bodies of the retina and cavernous atrophy of the optic nerve. Trans. Sect. Ophth., Amer. Med. Assoc., 1921, p. 146; Arch. of Ophth., 1922, v. 51, pp. 120, 239; 3 plates.

[4] Levine, J. Acquired cyst of optic disc. Arch. of Ophth., 1932, v. 8, pp. 209-217.

A SIMPLE METHOD FOR CALCULATING LENS POWER*

JOSEPH I. PASCAL, M.D.
New York

The usual methods of calculating the power of a lens involves the use of equations consisting of several fractions. The reason for fractional equations is the fact that the elements involved are related inversely; that is, as one element grows larger its related element grows smaller and *vice versa*. This inverse relation has to be expressed by means of reciprocals or fractional values. Fractional equations are rather cumbersome to work with, but, what is worse, the formula itself in fractional form is easily forgotten.

It is possible to calculate for lens power by a method that eliminates fractional equations and by a formula that is easily remembered and retained. This method involves the introduction of a *unit* of *curvature* corresponding to the *unit* of *power* we are all familiar with. Let the curvature of a surface having a radius of one meter (100 cm., 1,000 mm., 40 inches) be designated as one metrec (condensation of the words meter-curve). Then the relation of radius to curvature in *metrecs* is exactly the same as the relation of focus (focal length) to power in *diopters*. The focus of a lens, f, is related to its power in diopters, D, through the key numbers 1 (meter), 100 (cm.), 1,000 (mm.), 40 (inches). For example, if f = 20″, power = 2.00 D; if f = 40 cm., power = 2.50 D. If f = 20 mm., power equals 50.00 D., and so on.

Exactly the same relation through the same key numbers exists between the radius of the surface, r, and the curvature of the surface in metrecs, M. For example, if r = 20″, curvature = 2.00 M;

* Presented before the New York Society for Clinical Ophthalmology, May 1, 1939.

if r = 40 cm., curvature = 2:50 M; if r = 20 mm., curvature equals 50.00 M; and so on.

The power of the surface of a lens depends upon its curvature and the index of refraction, *n*, of the glass used. If *D* stands for the power in diopters, *a* stands for the *a*mount *a*bove 1.00 of the index, and *M* for the curvature in metrecs, then

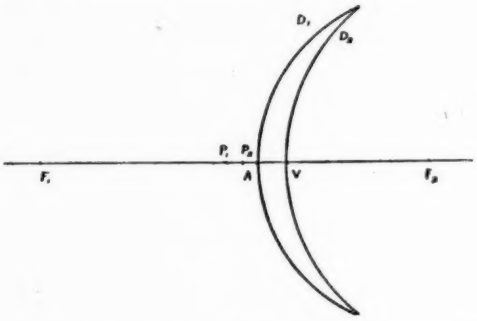


Fig. 1 (Pascal). Position of the principal and focal points, and the points of reference for the several focal lengths.

the power can be expressed by the formula D = aM; power = amount of index above 1.00, times curvature in metrecs. Half a dozen problems worked with this *Dam* formula will demonstrate the convenience of the method, and the ease with which the formula is remembered.

For example, what is the power of a biconvex lens of radii r and s, in which r = 10″ and s = 16″, made of a glass whose index (n) = 1.52? Take each surface separately. Radius 10″ gives M = 4; a = .52 (the *a*mount *a*bove 1.00 in the index). Substitute these in the Dam formula, and we get D = .52 × 4 = 2.08 D. The second radius s = 16″; M = 2.50; a = .52. Therefore D = .52 × 2.5 = 1.30 D. Both surfaces are convex, therefore the basic power is

	plus 2.08
	plus 1.30
total	plus 3.38 D

By the basic power is meant the sum of the surface powers. The modifications that have to be made because of thickness and other factors are minor and may best be regarded as refinements of the basic power.

Let us take another example, a biconcave lens in which the radii r and s are given as 20 cm. and 50 cm., and n = 1.63. By using the Dam formula and denoting the power of the first surface by D_1 and of the second surface by D_2, we have $D_1 = .63 \times 5 = 3.05$ D. and $D_2 = .63 \times 2 = 1.26$ D. Both surfaces are minus, therefore, basic power equals

	− 3.05
	− 1.26
total	− 4.31 D.

As one more illustration, suppose in a meniscus (contact lens) the radius of the convex surface equals 9 mm. and of the concave surface equals 8 mm; n = 1.516. Then $D_1 = .516 \times \frac{1000}{9} = \frac{516}{9} = + 57.33$; $D_2 = .516 \times \frac{1000}{8} = \frac{516}{8} = - 64.50$.

Basic power is

	minus 64.50
	plus 57.33
total	− 7.17 D

Of course the last problem could have been solved by first dividing 1,000 by 9 for D_1 and 1,000 by 8 for D_2 and then using the resulting number for M. But it is often easier to put the work in the manner indicated making the multiplication and division at the same time, so to say.

We may now consider briefly the relation of basic power to principal power (or true power), to apex power (or neutralizing power), and to vertex power (or effective power). Let us take a convex

meniscus lens, figure 1, in which $D_1 = +10.00$ D and $D_2 = -6.00$ D, thickness t = 3 mm., and index n = 1.52. In figure 1, F_1 and F_2 are the principal focal points; P_1 and P_2, the two principal points; A and V the apex and vertex of the lens, respectively. The lens as a whole has three focal lengths: (1) F_1P_1 which is equal to F_2P_2 and called the principal focal length; (2) F_1A, the apex focal length; and (3) F_2V, the vertex focal length. As was said at the beginning of the discussion, and as every doctor knows, the focal lengths f and D are related through certain key numbers, 1 (meter), 100 (cm.), 1,000 (mm.) and 40 (inches). But as there are three focal lengths that usually differ from one another, it is evident that there will be correspondingly three different powers. Let D_p stand for the principal or true power as determined by principal focal length, F_1P_1 or F_2P_2; let Da stand for apex (neutralizing) power as determined by the apex focal length, F_1A, and let Dv stand for vertex (effective) power as determined by the vertex focal length F_2v. Then the three formulas which express the three powers in terms of the surface powers D_1 and D_2, thickness and index, are as follows:

(1) $D_p = D_1 + D_2 - D_1D_2e$
(2) $D_a = D_1 + D_2 + D_2^2e +$
(3) $D_v = D_1 + D_2 + D_1^2e +$

It will be noticed that in all three formulas the first two terms give the *basic* power, the third term is the modification or trimming, as mentioned earlier in the discussion.

The *e* in the formula is the equivalent thickness, which is the actual thickness in *meters* divided by the index of refraction; namely $e = \frac{t(m)}{n}$, in which (m) means thickness measured in meters. In our example $e = \frac{0.003}{1.52} = 0.002$ approximately

$$D_p = +10 - 6 + 10 \times 6 \times e = 4 + 60e$$

$$D_a = +10 - 6 + 6 \times 6 \times e = 4 + 36e$$

$$D_v = +10 - 6 + 10 \times 10 \times e = 4 + 100e$$

It will be seen from the above that in this particular example, and in fact in all convex meniscus lenses, the true (principal) power, 4 + 60e, lies between the apex (neutralizing) power which is the least, 4 + 36e, and the vertex (effective) power, 4 + 100e, which is the greatest. When this third term is solved the result is

$D_a = 4 + .07$ D $= 4.07$ D
$D_p = 4 + .12$ D $= 4.12$ D
$D_v = 4 + .20$ D $= 4.20$ D

The effective power in this lens is about an eighth of a diopter more than its neutralizing power.

An examination of the third term in the equations, the term that modifies the basic power, will show some relationships that will make memorizing easier. The third term in D_p is minus the product of the surface powers D_1 and D_2 times e. If D_1 and D_2 are *both* plus or *both* minus the minus sign in the term remains minus. But if one of the two surface powers is plus and the other minus then the minus in the equation becomes plus as in our example (really the product of a minus by a minus, which thus becomes plus). Both surface powers enter into the term since the power is measured from the principal points. In Da, the third-term sign is always plus (the square of any quantity is always plus), and the product D_2^2e, can be written $D_2 \times D_2e$; that is instead of $D_1 \times D_2e$, we have D_2D_2e. The first surface (D_1) does not enter, its power is not modified, since the total power is measured from that surface; thus we have the second surface D_2 twice.

Likewise in D_v the third-term sign is always plus, and the product D_1^2e can be written $D_1 \times D_1e$. Here instead of D_1D_2e we have D_1D_1e. The second surface D_2 does not enter into the term since it is not modified, the total power being measured from the second surface D_2. Finally in the three formulas given, that for D_p is exact, that for D_a and D_v is approximate, but the approximation is so close that these formulas can safely be taken for all practical work.

37 West Ninety-seventh Street.

THE SIGNIFICANCE OF THE ORBIT LEVEL IN MUSCLE BALANCE

THOMAS H. ODENEAL, M.D.
Gloucester, Massachusetts

My investigation of this subject was to determine the effect of unlevel orbits on the ocular-muscle balance and to determine if such a condition affected the position in which the patient held the head.

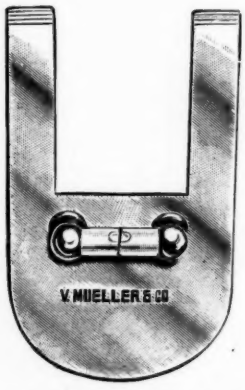


Fig. 1 (Odeneal). Orbit leveler.

An initial series of 200 cases was examined, the orbit level being determined by the first instrument* (fig. 1). The percentage of cases with unlevel orbits was so great, however, that it seemed certain that a considerable number were due to the position in which the head was held, although in each case the head was straightened as much as possible by using one arm of the orbit leveler. The same firm then made another instrument (fig. 2) that would be effective in bringing the patient's head to a level position. The cross-bar of the head leveler is placed across the vertex of the skull and the vertical bars, which are adjustable, are brought close against the sides of the head so that they are equidistant from the center of the cross bar, then the head is straightened until the water level is at center. The orbit leveler is now placed below the orbits with the ends of the arms at the margins, and the amount of unevenness in millimeters is read off when the water level is at center. The first instrument could be dispensed with by running a bar from one arm of the second

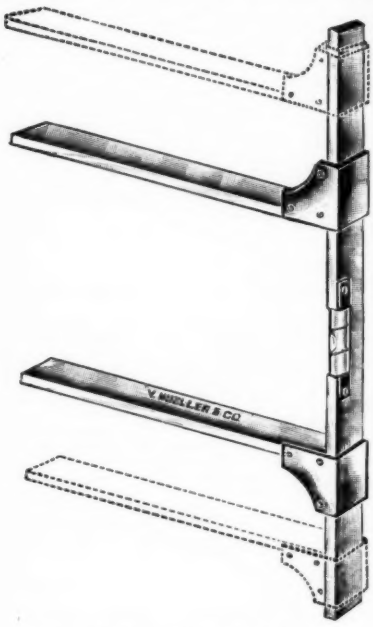


Fig. 2 (Odeneal). Head leveler.

* Made by V. Mueller & Company, Chicago, Illinois.

instrument to obtain the orbit level, but as the instrument was already available I continued to use it.

Each patient's muscle balance was measured with the lens correction in place, then the orbit level was found before and after the head was leveled, and the whole graded as follows: 1. Hyperphoria with unlevel orbits. 2. Hyperphoria without unlevel orbits. 3. Unlevel orbits without hyperphoria. 4. Normal, or level orbits without hyperphoria.

In a series of 89 cases the following was observed: 23 cases were in category 1; 18 cases were in category 2; 14 cases were in category 3; 34 cases were in category 4.

In this series, 14 cases presented uneven orbits without hyperphoria. This can be attributable only to the fact that the patient has been enabled through the years to adjust the muscle balance to the anatomical inequality of the orbits. It was necessary to prescribe prisms in only four cases of this series, since the others did not present symptoms attributable to imbalance of the muscles. Closure of one eye for several days caused hyperphoria in those patients with unlevel orbits who did not have hyperphoria before occlusion of the eye. (I cannot say that this is true in every case, as not all the patients with unlevel orbits had one eye occluded. It is difficult to get consent of the patient to go with one eye occluded for days.) This is to be expected in every case, for the muscles of the covered eye would naturally relax when it was no longer necessary for the two eyes to correct the inequality in their position.

If for some reason, such as lowered resistance, nervous exhaustion, and abuse of the eyes, the structure of the vertical muscles should be weakened, then hyperphoria would appear in those with uneven orbits, and this must be corrected if symptoms of muscle strain are present.

One thing that is rather disconcerting in this series is the fact that one third of the cases in group 1 presented a lower orbit level on the side of the hyperphoria. This must be attributable to overaction of the muscles in attempting to compensate for the inequality in position of the two eyes, causing a weakening of the opposing muscles.

As a great many people hold their head to one side or the other it was attempted to explain this by the unlevel orbits, but as some held their head to the side of the lower orbit and some to the side of the higher in group 3 this explanation was found of no value. Tilting the head to one side causes cyclophoria and not hyperphoria, and this is why patients with oblique-muscle trouble often tilt the head to one side.

The information gained in the investigation is evidence that unlevel orbits need not be taken into consideration in testing for muscular errors. From the results obtained in group 3 it is clear that the muscular system of the eyes takes care of this inequality, and when muscular error is present it should be corrected regardless of the orbit level. Also of value is the fact that patients with unlevel orbits without hyperphoria develop hyperphoria when one eye is occluded for several days. This fact must be remembered when attempting to find latent muscular error by occlusion of one eye for a time. When hyperphoria occurs on the side of the lower orbit, in the presence of associated symptoms, it should be corrected even though the original hyperphoria or tendency was indubitably present in the other eye at the onset.

MOVING-PICTURE RECORD OF THE ASTEROID BODIES IN A CASE OF ASTEROID HYALITIS*

JOSEPH WALDMAN, M.D.
Philadelphia

Asteroid hyalitis, although comparatively rare, is seen occasionally by almost every practicing ophthalmologist.

Benson,[1] in 1894, was the first to distinguish this condition from synchisis scintillans. Since then, cases have been reported periodically in the literature. In an excellent review, Rutherford[2] has carefully analyzed the cases reported up to 1933.

The outstanding condition from which asteroid hyalitis should be differentiated is synchisis scintillans. A summary of the principal characteristics of each follows:

ASTEROID HYALITIS. *Chemical composition:* According to Verhoeff,[3] "the balls consisted chiefly of a calcium soap, probably calcium margarate, possibly admixed with insoluble compounds of cholesterol and lecithin." Bachstez[4] concluded that the opacities consisted of fatty acid and lime, the fatty acid being either palmitic or stearic acid. Holloway and Fry,[5] in a series of microchemical tests, reported that "the spheres contain a carbonate, calcium, a stearate or a palmitate, or both, and probably lipoids in combination."

Ophthalmoscopic and slitlamp appearance: Examination of the vitreous reveals opacities occurring discretely or in clusters or strands, without any orderly arrangement. They are creamy, flat-white, or lardaceous as seen with the ophthalmoscope. The shape is usually globular. By transmitted light, they appear as dark droplets. The ophthalmoscopic picture has been graphically described as suggesting stars on a clear night, snowballs, snowball blossoms, or droplets of white paint suspended in water. But with the bright illumination of the slitlamp, they are more brilliant and seem to shine somewhat.

The viscosity of the vitreous is usually normal. When the eye moves, the opacities float with wavelike undulations. Usually they do not settle to the bottom of the vitreous but return to their former position more or less. Holloway and Fry found them as clusters in the extreme periphery when they were apparently absent from the body of the vitreous. He also stated they occasionally appeared in whitish streaks in the periphery resembling exudates.

SYNCHISIS SCINTILLANS. *Chemical composition:* In this condition, the vitreous opacities are usually cholesterol. In some cases tyrosine and leucine are suspected. This anomaly does not occur so frequently as does asteroid hyalitis.

Ophthalmoscopic and slitlamp appearance: As seen with the ophthalmoscope, the opacities in synchisis scintillans are more or less flat, angular, crystalline, irregular, and metallic in appearance. Ocular movements cause a rapid shower of these glittering, silvery, or golden bodies. They tend to settle quickly to the bottom of the vitreous. Dr. Alfred Cowan[6] describes them as showers of sparkling, bright coins. The vitreous is, as a rule, less viscous than in cases of asteroid hyalitis.

With the beam of the slitlamp, the characteristic appearance of these bodies is even more manifest.

Both asteroid hyalitis and synchisis scintillans should be differentiated from the following: (a) white globular opacities on a detached retina; (b) round corpuscular opacities in severe uveitis; (c) exudates in the periphery; (d) white nodes in the vitreous in retinoblastoma.

*From the Department of Ophthalmology, the Jefferson Medical College Hospital, Service 2.

J. L. Pavia,[7] in 1934, reported stereoretinographic and cinematographic records showing the movement and direction of the floating particles in a case of synchisis scintillans.

Moving-picture records of asteroid bodies were made from the left eye of a patient whose record follows:

J. F. F., a white male, 30 years of age, complained of a "mist in front of the right eye for the past week." His general health was good and he had never been seriously ill. There was a history of his having had "sinus trouble" three years previously but none since that time. He smoked 10 cigarettes daily and drank whiskey very rarely.

Ocular examination: The pupillary reactions, lid action, tension, rotations, and external examinations were normal. Vision with glasses was O.D. 6/35, O.S. 6/6.

The media of the right eye were clear; the disc was oval, its color, cup, edges, and vessel ratio were normal. Just above the fovea and involving the upper part of the macular region was a grayish-silver area slightly elevated, about one-half disc diameter in width and along its lower margin a reddish-brown hemorrhagic area with a moth-eaten appearance.

The vitreous of the left eye showed asteroid hyalitis; the disc was oval, its color, cup, edges, and vessel ratio were normal, as was also the macular area; no exudates nor hemorrhages were seen.

Slitlamp examination: O.D. normal; O.S., asteroid bodies were seen in the vitreous (fig. 1).

Visual-field study: O.D., the peripheral field was normal; there was an absolute paracentral scotoma. O.S., normal.

Physical examination was entirely negative for pathology.

Laboratory studies: X rays of the sinuses were negative; the blood Wassermann and Kahn tests were negative; the blood sugar, N.P.N., and cholesterol, normal; urinary studies, negative. There was no asthma, hay fever, nor food sensitivity.

The patient was very much surprised when he was informed that he had some abnormality in his left eye. As is well-known, the asteroid bodies in themselves cause no diminution of vision.

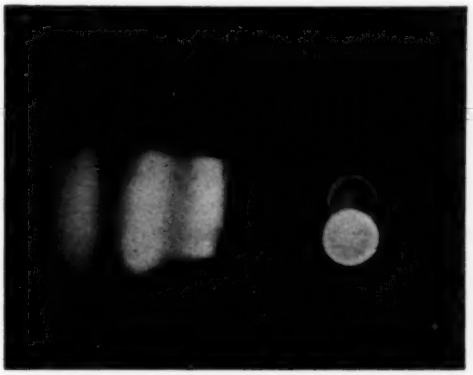


Fig. 1 (Waldman). From left to right, narrow white area is the cornea, black area is the anterior chamber, large white area is the crystalline lens, spherical white areas are the asteroid bodies in the vitreous.

Technique

The pictures were taken in a dark room. The patient was seated on a chair with his head and chin held in position on the ordinary slitlamp–corneal-microscope table. The pupil was dilated with one drop of 5-percent homatropine hydrobromide and 1-percent Benzedrine (amphetamine).

The corneal microscope was replaced by an Eastman Kodak special moving-picture camera with the small extension tube of the telescopic arrangement. This was placed on a small wooden platform of sufficient height so that the objective was on a level with the patient's eye. The objective was within an inch of the eye. Very slow and careful focusing is essen-

tial. Illumination was supplied by the ordinary slitlamp beam.

Super XX film was used. Color film was not used because the light was not bright enough.

The lens was set wide open at f 1.9. Various speeds of exposure were tried but 16 frames per second was found to be the most satisfactory.

At the present time, we are trying to use color film with the aid of a carbon arc lamp, the beam being cooled by a cylinder of water.

The above method may be applied to the study of the normal and abnormal vitreous body.

Conclusions

The film record shows that the asteroid bodies are spherical and occur singly. They may gather in small groups but do not coalesce or unite.

Through the view finder of the camera the spheres were of a pale yellow or dull golden color. They appeared translucent. The apparent difference in size of the opacities was due to their varying distances from the objective. Those that appeared to be on the same vertical plane were approximately of the same size.

The speed with which they moved was that of small vitreous opacities in a vitreous of apparently normal viscosity. There was no tendency to settle to the bottom of the vitreous chamber when the eye was not moving. On the contrary, they seemed suspended in the vitreous.

My appreciation for the assistance of Mr. Thomas Stewart, of Street, Linder, and Propert Company of Philadelphia, is gratefully acknowledged.

1930 Chestnut Street.

References

[1] Benson, A. H. A case of monocular asteroid hyalitis. Trans. Ophth. Soc. U. Kingdom, 1894, v. 14, p. 101.

[2] Rutherford, C. W. Asteroid bodies in the vitreous. Arch. of Ophth., 1933, v. 9, Jan., pp. 106-117.

[3] Verhoeff, F. H. Microscopic findings in a case of asteroid hyalitis. Amer. Jour. Ophth., 1921, v. 4, March, p. 155.

[4] Bachstez, E. Ueber den klinischen, chemischen, und histologischen Befund bei der Scintillatio corpora-vitrei. Zeit. f. Augenh., 1925, v. 54, p. 26.

[5] Holloway, T. B., and Fry, W. E. Microchemical and histological findings in a case of asteroid hyalitis. Trans. Amer. Ophth. Soc., 1929, v. 27, p. 165.

[6] Cowan, A. Personal communication.

[7] Lijo Pavia, J. Synchisis scintillans stereoretinographic and cinematographic records showing movement and direction of floating particles. Rev. Oto-Neuro-Oft., 1934, v. 9, March, pp. 93-97. Abstr. Bull. Soc. d'Opht. de Paris, 1934, Jan., pp. 35-36.

SOCIETY PROCEEDINGS

Edited by Dr. Rommel Hildreth

COLLEGE OF PHYSICIANS OF PHILADELPHIA

Section on Ophthalmology

January 19, 1939

Dr. Alexander G. Fewell, *chairman*

Anterior Cerebellar-Bulbar-Pontile Poliomyeloencephalitis; Final Ocular Result in a Case 27 Years After the Acute Attack

Dr. Edward A. Shumway said the patient, a woman 33 years of age, was admitted to the University Hospital in September, 1911, on the service of Dr. Charles K. Mills, at that time Professor of Neurology in the University of Pennsylvania.

The neurological examination showed complete facial paralysis on the left and hemiplegia of the right side. The tongue deviated to the right, and the jaw to the left. There was paralysis also, of the fifth and sixth cranial nerves, and of the associated movements of the eyes, both to right and left, and up and down.

Eye examination showed slight reduction of vision, but entirely normal eyegrounds.

Dr. Mills's final diagnosis, as the result of observations extending over years, was "Destructive poliomyelitis, cerebellar-bulbar, and pontile."

For a time the presence of tumor was considered, and operation at one time discussed and dismissed. The patient had been under Dr. Shumway's care in his private practice for 27 years.

As a result of the paralysis of the sixth nerve, which with the seventh-nerve involvement remained permanent, and the weakness of the associated movements both lateral and vertical, right hyperphoria and esotropia had developed, and increased from two degrees to six degrees, as a result of the turning upward and inward of the left eye. Esophoria had decreased from eight to six degrees. After the final stabilization of the muscle condition, the patient had been comfortable with the use of three-degree prisms, base up in the left eye and base down in the right eye, for distance, and prisms of two degrees in the same positions, in each eye, for reading. The esophoria did not need correction, and the patient was free from diplopia, in both far and near vision.

The final report of the neurological condition, by Dr. J. W. McConnell, made a few days before the meeting, showed absence of all sensory paralyses, recovery of the right hemiplegia except for some hyperactivity of the deep reflexes on the right side, permanent left facial paralysis which had been partly relieved by suturing the eyelids at the outer canthus. Finally as a remaining bulbar sign there was a paresis of the left half of the vocal cords, which was confirmed by Dr. Ralph Butler, and was due to motor disturbance of the left recurrent laryngeal nerve.

Discussion. Dr. Walter I. Lillie said that Dr. Shumway is to be envied in having had the privilege of observing a case over a period of 27 years. It does not fall to one's lot to be able to do this often. This is obviously a case of poliomyelitis, because of its history and the fact that the residual defects are entirely motor. Localization is obviously correct because it included DeFoville's syndrome, which is paralysis of the sixth and seventh nerves with a contralateral hemiplegia. The patient also had a homolateral Horner's syndrome, which fits into this picture. It has

always been interesting that these patients do not have involvement of the eighth nerve, in spite of the fact that auditory nuclei are situated in the vicinity of the lesion. He believes that if the hearing could be examined like the visual fields, a homonymous hearing defect would be found in conditions of this sort.

Dr. Shumway, in conclusion, said that the patient's hearing on the left side, is now four tenths of normal, and that this deafness was due to middle-ear involvement, probably a former suppurative inflammation, as there is a scar on the eardrum. Deafness may have been due to involvement of the eighth nerve, however, as Dr. Lillie had suggested. He also corroborated Dr. McConnell's findings, the paresis of the left half of the vocal cords, which is the sole remaining sign of the bulbar involvement.

His reasons for presenting this case were, in the first place, to report the ability to make the patient comfortable from an ophthalmological viewpoint by vertical prisms, and also to stimulate interest among ophthalmologists in neuro-ophthalmology.

Adie's syndrome

Dr. Harold G. Scheie presented a case of the complete form of Adie's syndrome. The importance of care in recognizing the condition and differentiating it from tabes dorsalis with Argyll Robertson pupils was emphasized. A theory as to the mechanism of its production was suggested. Since the pupil contracted well to eserine and was sensitized to choline compounds it was felt that a partial denervation of the sphincter might have occurred.

Discussion. Dr. Francis Heed Adler said that Kinnier Wilson made the statement that no one should make a diagnosis of Argyll Robertson pupil unless he had read Argyll Robertson's original paper. It is certain that many pupils are wrongly diagnosed as Argyll Robertson pupils because all of the characteristics of this phenomenon were not strictly adhered to. Adie's syndrome is a case in point. We cannot be certain at the present time where the site of the lesion is in Adie's syndrome, but with the help of animal experimentation, it is possible that Dr. Scheie will be able to locate the lesion more accurately.

Dr. Scheie, in conclusion, said the patient presented tonight has no symptoms, because only the ciliary muscle of one side was affected. If both sides were affected, the patient would certainly complain of blurring when changing the distance of gaze.

This patient was tested before and after the use of choline, but he found little or no change from its use.

The various types of eye movements and their clinical significance

Dr. A. Bielschowsky, Hanover, New Hampshire, stated that there are four main groups of eye movements: the voluntary (commanded) movements, the attraction movements, which are brought about by sensorial stimuli attracting attention, the fusion movements, and the reflex movements of vestibular origin. The second and third groups are called psychoöptic reflexes because they take place almost automatically, without awareness of intention on the part of the individuals, but are not true reflexes in as much as they are elicited by sensorial stimuli only if the latter attract attention.

Through error this strict validity of the law, according to which under normal conditions every innervation flows equally to both eyes, has been doubted. The fact that a single eye can be moved separately does not prove that the unilateral movement is produced by a uni-

lateral innervation. If two impulses are sent to the eyes simultaneously, one driving them in the same direction, the other in the opposite one, an unequal or even unilateral movement must result. An eye movement is never brought about by one single muscle. All the ocular muscles always coöperate in performing any ocular movement, one half acting as agonists, the other as antagonists. The same cortical center that controls the contraction of the agonists brings about the active relaxation of the antagonists, so that a movement may take place even if the agonists are unable to obey this motor impulse because of a total paralysis.

The peculiarities of fusion movements were discussed, and their importance for the visual act, particularly in people with heterophoria, which can be kept latent only as long as the fusion mechanism maintains the innervation compensating the anomalous position of the eyes relative to each other.

The only true reflex movements of the eyes originate in the vestibular apparatus. Every change of the position of the head or body causing a current of the endolymph in the semicircular canals, causes a stimulation that is conveyed to the motor nuclei by the posterior longitudinal bundle. The reaction of the ocular muscles aims at keeping the position of the eyes in space unaltered, notwithstanding the change of the position of the head or body.

A characteristic vestibular reaction is presented by the parallel rotary movement of the eyes around their anteroposterior axes, elicited by the tilting of the head toward the opposite side. This reaction is at the bottom of ocular torticollis which, in cases of trochlear palsy and other disturbances of the oblique muscles, helps the patient to see single. The head tilting test (Bielschowsky and Hofmann) is described as a diagnostic method in disturbances of the vertical motors of the eyes.

The reaction of the eyes to vestibular stimuli is most important in cases of supranuclear palsies. If the muscles do not respond to voluntary impulses while they react to vestibular stimuli produced by Bielschowsky's head-rotation test or Barany's test, this is proof that the nuclei and the peripheral nerve fibers are intact. The characteristics of attraction and following movements are discussed with regard to those cases of supranuclear palsies in which only the following movements are obtainable. The importance of Bell's phenomenon is illustrated by reports of cases with paralyses of the associated upward movement, where Bell's phenomenon was the only proof of the supranuclear site of the lesion. According to the particulars, which can be ascertained by the various methods of investigation, it may be possible to make an approximate localization of the lesion causing the associated paralysis in an individual case.

Warren S. Reese,
Clerk.

NEW YORK EYE AND EAR INFIRMARY CLINICAL CONFERENCE

January 30, 1939

DR. SIGMUND A. AGATSTON, *chairman*

CASE FOR DIAGNOSIS

DR. ARTHUR C. CHANDLER presented a case for diagnosis. The patient, a female of 37 years, with evidence of a severe rachitic condition and shortening of the legs, accompanying stature deformity, was first seen in November, 1937, complaining of itching, tearing, and slight pain in the eyes. The condition had been present for 18 months, being worse in the right eye. The conjunctiva was

congested and gave the appearance of vernal catarrh of the bulbar type. For two months there had been a persistent purulent discharge which was very likely a secondary infection. Vision was unimpaired according to the patient's statement. She gave a history of asthma for the past 10 years, being more marked in September and October. During these attacks the eyes become red and swollen.

X-ray studies of the skull and sinuses showed nothing abnormal; head, ears, and nose were normal. The eyes showed a purulent discharge with red and thickened conjunctiva; there was no pus in the sacs. Vision O.D. was 20/70; O.S. 20/40. Wassermann reaction was negative. No eosinophiles were found in any of several smears from the conjunctiva and mucous membrane of the nose. Smears showed staphylococcus and extracellular diplococci in pairs and chains, and pus cells. A diagnosis of vernal catarrh of the bulbar type was made. Treatment was local, consisting of application of mercurochrome and silver nitrate. On December 6, 1937, she was seen by an allergist, who made a diagnosis of perennial vasomotor rhinitis after finding her sensitive to dust, flaxseed, and numerous food articles. Desensitization therapy was instituted. A polyvalent catarrhal vaccine was given.

She was discharged from the hospital in December, 1937, her condition being improved. Local treatment was continued, consisting of zinc, boric-acid compresses, and even copper for a short time, but the right eye continued to become progressively worse. There was some congestion of the bulbar conjunctiva and episclera on the temporal side of the left eye and tenderness in the vicinity of the insertion of the lateral rectus muscle. In November, 1938, a Mantoux test was strongly positive. Atropine was begun and T.R. dil. No. 1 started, but after three or four injections this was discontinued when she was admitted to the hospital with a diagnosis of lattice keratitis. Vision O.D. was 5/200 at this time. Ultraviolet irradiation of one minute daily, gradually increasing to three minutes was given for one week. Vision continued to fail. Mercury biniodide was started and continued for about two weeks until the patient was discharged to the clinic on December 24, 1938, with vision in the right eye of light perception. Four intravenous administrations of typhoid vaccine were given.

On January 10, 1939, the patient was readmitted to the hospital. The lids were red and the patient seemed very uncomfortable with pain, photophobia, and lacrimation. There was ciliary tenderness and the eye was very red and soft. Descemet's membrane was wrinkled and there was marked interstitial involvement. The pupil was small and efforts to dilate it failed, and, of course, the fundus could not be seen at this stage. On January 17, 1939, the right eye was removed. A few days after enucleation, the patient complained of pain and tearing of the left eye. This has improved somewhat, but there remains some redness and congestion of the conjunctiva and of the episclera of the temporal side of the eye. The fundus shows no pathology. Vision is about 20/30. She has been in the hospital and has been receiving homatropine t.i.d., and T.R. dil. No. 1 twice weekly. Their problem is to arrest the condition in the left eye to prevent total loss of vision.

On section the enucleated globe showed no evidence of tuberculosis.

Discussion. Dr. Clyde E. McDannald said that in vernal catarrh there is seldom corneal involvement. He added that Dr. Brown Pusey called attention to the relationship between vernal catarrh and allergy 35 years ago.

Dr. Sigmund Agatston agreed with Dr. McDannald as far as allergy is concerned, believing vernal catarrh and allergy to be associated. Possibly choroiditis, which is supposed to be a focal manifestation, may be an allergic manifestation, and this patient may have had an allergic manifestation in front as well as posteriorly.

Epithelization in the anterior chamber following cataract extraction

Dr. Arthur C. Chandler said that this patient had had a modified Lagrange operation for chronic simple glaucoma in September, 1938. On December 13, 1938, he had an extraction of the right lens after preliminary iridectomy. His convalescence was uneventful, but he had some redness of the eye on discharge. On January 3d ciliary injection was noted, and on the fifth it was noticed that a small gray membrane was growing down over Descemet's membrane at the 11-o'clock position. This has increased in size until it now covers one third of the cornea, extends back, and is attached to the iris. Dr. Perera, Dr. Chandler said, has reported a case of epithelization of the anterior chamber which was cured by radium. The patient was referred to the Radium Clinic.

Melanosarcoma of the ciliary body

Dr. Samuel Gartner reported that the patient, a 27-year-old woman, gave a history that the day before coming to the clinic the vision in the left eye became poor. She did nothing about it that day, and the next morning her vision was better. She was walking up stairs the day she appeared at the clinic, when her vision suddenly went bad. Vision was 20/70 O.S.; 20/30 O.D. when first seen. The right eye was perfectly normal, but the left eye externally showed the conjunctiva to be pale, except above the limbus where it was flushed with dilated blood vessels. The cornea was clear; the iris very striking. The pupil was displaced below the center, the upper border was flattened. The upper part of the iris bulged forward against the cornea. In the pupillary area, between the iris and lens, there was a small dark-brown mass. The lens was clear, the vitreous hazy. No abnormality of the fundus could be made out. Tension was normal. The field of vision in the left eye showed a large defect below and temporally. The right eye was normal. Transillumination of the left eye showed a positive shadow above in the region of the ciliary body extending from 9 mm. behind the limbus to the limbus. Urinalysis and Wassermann reaction were negative. Diagnosis of melanosarcoma of the ciliary body was made. Dr. Agatston enucleated the left eye.

Discussion. Dr. McDannald said that from the clinical standpoint and from the history given, this type of case develops rapidly. Dr. Boyes and Dr. McDannald had a case that they watched for four years before it was determined that the woman had a sarcoma of the ciliary body. After enucleation the diagnosis was confirmed. Another case, the patient a 24-year-old woman, was also followed for four years before a definite diagnosis of sarcoma of the ciliary body was made. Dr. McDannald thought that these tumors are apt to be rather slow in developing.

Dr. T. L. Boyes said that Dr. McDannald's patient was in her early twenties, and his patient was over 60 years old. He watched her for five or six years after enucleation, and she was well. He added that these cases are slow in coming to any point of decision, but eventually they develop secondary glaucoma. The only clinical symptoms in Dr. Boyes's case were a slightly pear-shaped

pupil and diminished transillumination. He added that it is difficult to persuade these patients to have the eye enucleated since they have good vision and no pain.

Dr. Donald W. Bogart described an interesting case of sarcoma of the ciliary body. The diagnosis had been made by other ophthalmologists. The patient, a boy in his early twenties, had agreed to have the eye removed. Gonioscopic examination showed that the tumor had depressed the iris and the iris process stretched out, but the angle was open. That may have had an effect on increasing drainage. The eye was enucleated, and four weeks later the boy was taken back to the hospital definitely moribund. The patient died. He was found to have many metastatic nodules in the skin. The question arises whether this tumor of the eye was primary or secondary.

Dr. Agatston expressed the opinion that sarcoma of the ciliary body is more dangerous than sarcoma of the choroid. He added that there is a way of determining metastases early in the liver by endoscopy. In a case he had in which a diagnosis of sarcoma of the choroid was made, this test was attempted, but no cells were found in the liver. Dr. Agatston then told of a patient who had come to see Dr. Nathan Cohen. An iris bombé with a protrusion that looked like a tumor was present, hence diagnosis of tumor was made. Transillumination was positive. On operation it was found to be a cystic condition with typical iris bombé. Four months earlier the patient noticed gradual loss of vision. When Dr. Cohen saw the patient, no light perception was present. The eye was hard, the cornea clear, the iris bulged forward; the pupil was small, the margin bound down to the lens. Transillumination in the area where the iris bulged was positive. The patient was seen that night at a meeting of the Academy of Medicine, and was examined by about 90 ophthalmologists who agreed that it was a case of melanosarcoma. The eye was enucleated.

Solar retinitis

Dr. Paul McAlpine reported that the patients, two brothers, had purchased a sun lamp and sat before it for two 15-minute intervals without glasses. That evening photophobia and lacrimation were present. The next day they were seen at this hospital with stippling of the cornea, conjunctivitis, and extreme photophobia. The next day they were hospitalized. By the following day the conjunctivitis had subsided. There was definite edema of the macular area in one boy's eye. The other eye could not be seen. Vision was limited to the ability to detect hand movements. Fields taken on the older brother were limited to a tiny crescent. The younger boy's fields were practically the same, but the field was a little larger. The condition remained essentially the same for almost a week although the conjunctivitis and keratitis cleared up in a few days. The retina shows essentially nothing. Blood counts were normal; sinus X-ray films negative; medical examination negative. Subsequently the eyes cleared entirely.

Juvenile glaucoma with staphyloma

Dr. John Cregar reported that this girl was sent from the South three years ago with a history of good vision (about 20/30), but the fields were contracting. Examination showed she had 20/30 vision O.U. Her accommodation was about the same as that of a person 50 years old. She could not read without a +2.00 D. lens in front of each eye. Her fields were cut down concentrically. She had no unusual nasal difficulty. Bilateral iridocorneo-sclerectomy was performed with no incarceration of the iris. About two

months ago she returned with vision O.D. of light perception; O.S. 20/50. In order to get 20/50 vision in the left eye a +7.00 D.cyl. had to be used. At the site of the operative wound in the left eye she had two blebs that resembled staphylomas. The right eye had developed a cataract. The tension was normal in both eyes. Both pillars were incarcerated. She began to run a temperature at this time, and a medical check-up showed that she had appendicitis. She was removed to a general hospital; her eye condition was not improved. She had much pain and developed an acute ectasis of the eye that night, when a pressure bandage was put on the eye, and a little atropine instilled. The next day the eye was about the same. Her appendix was removed and was found to be normal. The cornea then began to cloud and paracenteses were made. The eye was opened and a prolapse of the iris into the old wound was found. The prolapse was excised and the iris incarcerated in the lips of the wound, and conjunctiva pulled over. Vision was light perception, and tension was normal. The other eye has a mature cataract.

Retinitis striata

Dr. Donald Hall reported that the patient, a 24-year-old girl, gave a history of having been in an inconsequential automobile accident. One week later she noticed that the vision in the left eye was blurred. On admission there was found to be a flat detachment of the lower part of the retina with two large streak exudates. A diagnosis of flat retinal detachment with striped retinitis was made. A focal-infection study revealed no infective foci. Altitudinal hemianopsia was present in one eye. Dr. Hall questioned whether this condition is due to trauma or infection, and asked what should be done for this patient.

Retinitis pigmentosa

Dr. Donald Hall said that this patient gave a history of no vision in the affected eye for 20 years and for the past few weeks the vision in the other eye was becoming blurred. A complete study for foci of infection showed nothing. The blood Wassermann was negative. The family history showed nothing from the standpoint of retinitis pigmentosa. The fields did not show ring scotomata. The patient has two brothers with poor vision, and his three sisters have good vision. The parents have no trouble with their eyes. He had an old luetic condition and had received antiluetic treatment.

Discussion. The consensus of opinion was that the case was one of disseminated choroiditis.

Donald W. Bogart,
Secretary

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Section on ophthalmology

February 10, 1939

Dr. Erling Hansen, *presiding*

Surgical and applied anatomy of the eye and its adnexa

Dr. F. Bruce Fralick of Ann Arbor (by invitation) took up the clinical points of interest in the anatomy of the orbit. He stressed the importance of implanting a solid substance like gold or glass in Tenon's space following enucleations performed during the period of orbital growth. The orbit conforms to the law of adaptation of the organ to the function which it is called upon to fulfill, and if the globe be removed before the orbit is fully formed, growth of the latter is checked, resulting in more or less facial asymmetry.

The weak areas in the orbital walls were pointed out in order to draw attention to the possibility of perforating these walls in performing exenterations, which would result in meningitis if the superior wall were perforated, or a proliferation of the sinus mucous-membrane lining to give a weeping mucous-membrane lining to the orbit rather than a dry cutaneous surface if the nasal wall were perforated. The paper plate of the ethmoid is a likely avenue of dissemination of inflammation to the orbit, thus producing orbital cellulitis, mucocele, or phantom tumor.

The facial planes of the orbit were next considered, and it was pointed out that when the globe is removed Tenon's space has a diameter of not greater than 18 mm. in the adult and less in the child, for receiving a glass or gold-ball implant. A larger ball is likely to be pushed through the posterior opening in the capsule that is produced when the optic nerve is severed, thus causing it to migrate into undesirable positions in the orbit, or to result in a higher percentage of extrusion than is necessary.

In taking up the anatomy of the eyeball, Dr. Fralick pointed out that the sclera has relatively few blood vessels except for an extremely attenuated episcleral network and a denser plexus near the corneal margin. The scarcity of blood vessels and the histological structure of the sclera account for the slow healing and gaping of scleral wounds. The thickness of the sclera was emphasized as being of importance in recession operations in that here it varies from 0.3 to 0.5 mm. in men and is thinner in children and women.

The relatively few anastomoses between the choriocapillaris anterior and posterior to the equator, which is supplied by recurrent branches of the long posterior ciliary arteries and short posterior ciliary arteries, respectively, have been cited as the cause of failure of the circulation of the intermediate zone in retinitis pigmentosa. However, the ring scotoma in retinitis pigmentosa is situated between the 20- to 30-degree meridians, which is in the zone of the postciliary circulation and not at the zone of anastomosis. Ring scotoma at the equator is seen in syphilitic diseases of the arteries and may be used as a differential point.

The suprachoroidea becomes lost in the meridional part of the ciliary muscle, so that there are no suprachoroidal lamellae in the anterior part of the ciliary body. Because of the density of the lamina and the absence of suprachoroidea beneath at least a part of the width of the ciliary muscle or body, it was suggested that in performing a cyclodialysis better results would be obtained if the dissection is carried behind the scleral opening, thus opening a passage into the subchoroidal space behind the ora.

The anatomy of the pars planum of the ciliary body was reviewed, and it was pointed out that there is no choriocapillaris and that the vessels in the pars planum are largely meridionally coursing veins which run back to the vena vorticosa. Dr. Fralick uses this area as the safest approach for the removal of magnetic intraocular foreign bodies. A hinged trephine button of the sclera over the pars planum is made and the pars planum incised meridionally, parallel to the veins in this area. After the foreign body is removed, the scleral button is replaced, and a double layer of conjunctiva is sutured over it. Very little reaction follows this approach.

The thickness of the lens capsule varies in different portions and increases in thickness throughout with advancing years. It is thickest in a zone running around concentrically with the equator and a short distance axial to the insertion of the zonular fibers on both the anterior

and posterior lens surfaces. Since the zonular fibers weaken with age, and the capsule of the lens thickens with age, we have a reason for the greater ease of intracapsular extraction in elderly people. Possibly by grasping the capsule in its thickest portion, we would rupture the capsule less often.

The importance of remembering the length of the optic nerve in performing external ethmoid operations was stressed in an attempt to prevent the development of optic atrophy from too much tension being placed on the nerve when exposing the ethmoid area. The average length of the optic nerve between the globe and optic foramen is 25 mm., while the average distance between the posterior pole of the eye and the optic foramen is 18 mm., thus allowing a possible forward displacement of the globe of 7 mm.

The length of the extraocular-muscle tendons was considered of clinical importance in resection operations in that there are statements in the literature advising as much as 12 mm. of resection of muscle tendons. Obviously this is impossible in that the longest rectus tendon is that of the external rectus, which is 8.8 mm. long (medial rectus 3.5 mm., inferior rectus 5.5 mm., superior rectus 5.8 mm.). By removing more than tendon we are performing a myectomy, thus defeating the purpose of the shortening operation.

The physiological pull of the superior and inferior recti was illustrated, showing that O'Connor has rightly pointed out that in performing a transplantation of superior and inferior recti to augment the abductor action of a paretic externus, we should transplant the nasal halves of these muscles rather than the temporal halves, thus throwing the maximum amount of pull temporal to the vertical axis of rotation of the eyeball.

The blood supply of the conjunctiva was reviewed. This is basic for a clear understanding of the different types of injection given clinically for conjunctivitis, iritis, superficial corneal lesions, and interstitial keratitis.

The venous drainage of the orbit was illustrated, showing the possible paths by which the blood could drain in cavernous-sinus thrombosis. It was pointed out that the central vein of the retina opens most frequently separately into the cavernous sinus, rarely into the superior or even inferior ophthalmic vein, but always has at least one side connection. This connection is commonly with the superior ophthalmic vein, so that even in the presence of cavernous-sinus thrombosis we do not see the fundus picture of occlusion of the central retinal vein.

The relations of the internal palpebral ligament were illustrated, attention being called to the danger of approaching the posterior lacrimal crest too closely in operations on the lacrimal sac, in that if the tensor tarsi (Horner's muscle) is severed, epiphora will result through eversion of the puncti. Paralysis of Horner's muscle in facial paralysis results in the earliest symptom of this condition; that is, epiphora. The thinness of the upper part of the anterior division of the internal palpebral ligament and its attachment to the lacrimal fascia over the upper portion of the lacrimal sac affords a possible explanation of cases in which the lids swell up on blowing the nose after a comparatively light blow to the eye; a sudden strain being thrown upon the ligament so that the sac is torn.

The abnormalities seen in the lacrimal duct may account for the inability to probe in stenosis at times. In congenital stenosis of the lower end of the nasolacrimal duct, it is sometimes necessary to incise the nasal mucous membrane over the passed probe, due to the fact that the true opening, although closed, is

higher than the lower end of the duct. In these cases the probe reaches the floor of the nose but, when removed, fluid cannot be irrigated into the nose, indicating that no opening has been made into the nose, but that a blind passage has been found.

Discussion. Dr. Fralick said in answer to Dr. J. J. Prendergast that he did not cauterize the uveal tissue before reinserting the scleral button after foreign-body operation. There is practically no bleeding, and since there is no danger of retinal detachment at this area, he has not seen the need for this procedure.

George E. McGeary,
Secretary.

COLORADO OPHTHALMOLOGICAL SOCIETY

February 18, 1939

Dr. Edward Jackson, *presiding*

The following motion pictures were shown: Jameson recession operation, Tenotomy of the inferior oblique muscle by Dr. William P. Beetham, Intracapsular cataract extraction by Dr. Beetham, and Keratoplasty by Dr. Ramon Castroviejo.

Congenital melanosis of the retina

Dr. James Shields presented the case of Mr. R. S., aged 30 years, who had consulted him on account of severe headaches. The patient's general health had been good except for chronic osteomyelitis of the hand. The eyes were normal in every way except for a mild pigmentation of the left fundus. The vision was not affected. Dr. Shields stated that the condition was probably the result of congenital hyperplasia of the pigment cells of the retinal epithelium.

Conjunctival hemangioma

Dr. William M. Bane presented the case of J. H. M., aged 42 years, who had consulted him because of persistent redness of the conjunctiva of the right eye. A red elevated mass had been present in the bulbar conjunctiva temporal to the limbus since 1934. The left eye was divergent and amblyopic. A diagnosis of hemangioma of the conjunctiva of the right eye was made.

Discussion. Dr. Melville Black recommended that the hemangioma and surrounding conjunctiva be surgically excised and the resulting conjunctival defect be closed by suturing the conjunctiva together.

John C. Long,
Secretary.

AMERICAN JOURNAL OF OPHTHALMOLOGY

Published Monthly by the Ophthalmic Publishing Company

Address original papers, other scientific communications including correspondence, also books for review and reports of society proceedings to *Dr. Lawrence T. Post, 640 S. Kingshighway, Saint Louis.*

Exchange copies of medical journals should be sent to *Dr. William H. Crisp, 530 Metropolitan Building, Denver.*

Subscriptions, applications for single copies, notices of change of address, and communications with reference to advertising should be addressed to the *Manager of Subscriptions and Advertising, 640 S. Kingshighway, Saint Louis.* Copy of advertisements must be sent to the manager by the fifteenth of the month preceding its appearance.

Author's proofs should be corrected and returned within forty-eight hours to the *Manuscript Editor.* Twenty-five reprints of each article will be supplied to the author without charge. Additional reprints may be obtained from the printer, the George Banta Publishing Company, 450-458 Ahnaip Street, Menasha, Wisconsin, if ordered at the time proofs are returned. But reprints to contain colored plates must be ordered when the article is accepted.

THE SOUTHERN MEDICAL ASSOCIATION MEETING IN MEMPHIS

Not farther than an overnight train trip from almost anywhere in the territory of the Southern Medical Association—and travel by rail has certainly become more alluring in the past few years—lies Memphis, host city of the Association for 1939. The meeting was held in the second Thanksgiving week, but seemingly this did not interfere with the attendance, which was very good. It happened not to be the week that Tennessee was celebrating, so the local men were not too busy to attend in goodly numbers.

In accordance with the usual custom, Tuesday was set apart for a presentation by local physicians. This was a particularly good program. To select one paper from the group for especial comment is difficult, but because of its timeliness and the fact that it was possible to see some of the reported cases on the following days, McKinney's description of 21 cases of corneal transplant was of particular interest (to be published in an early issue of the Journal). This is the kind of work that should at present probably be left to one or two men in the larger centers, for cases are rare and the technique is difficult. The results are perhaps not such as would be classed as brilliant, but in favorable cases do offer a chance of improvement that seems well worth while,

and certainly the subject should be pursued further.

Wednesday was delegated Ellett Day in honor of Dr. Edward C. Ellett, the erudite and charming Dean of Ophthalmology in the South, and a special program was presented in his honor. It was the best thing of its kind that the editor has ever attended. Dr. Walter Parker, a classmate of Dr. Ellett in medical school, who had come from Detroit to be present at this celebration, reminisced of student and intern days. He gave extremely interesting side lights on medical education of 50 years ago and told of Dr. Ellett's remarkable ability as a student, having maintained an unbelievably high standing epitomized by the story of how it was the habit of professors in his class when other students could not answer the questions to turn to Dr. Ellett and say, "You tell them Ellett." Harvey Searcy, one of his associates in times past, introduced a lighter tone by relating some amusing stories of this association. Grady Clay recited war experiences under his "colonel," and James Stanford told of what Dr. Ellett had done for ophthalmology in Memphis. Dr. M. M. Cullom, who presided, closed with a beautiful tribute.

The regular program was featured by talks from the two guest speakers, Dr. Harry Gradle and Dr. Chevalier L. Jackson. The former gave a practical talk on the treatment of glaucoma, illustrating by his failures some errors that might be avoided and thence traversing to a discussion of present techniques; the latter on treatment of cancer of the larynx. This has been a favorite subject of the speaker for many years and he gave a clinical talk beautifully illustrated with many colored slides.

Not having a great interest in otolaryngology, the editor listened to only a few papers on that subject. A very interesting contribution was that of Dr. John Bordley who reported having treated with marked success by implantation of radon seeds cases of deafness due to pathology at the outlet of the Eustachian tube, the particular cause for which was granulation of the scar tissues following improper or inadequate removal of adenoid tissue.

The entire program seemed well rounded and interesting. The tribute is for the secretary, not for the chairman, who had little hand in formulating it.

Louisville was chosen for the next meeting with Dr. Harry Searcy as chairman and Dr. Albert Leonard Bass as vice-chairman.

Lawrence T. Post.

THE ISOLATED STUDENT

A university course is more than an opportunity to read for examinations and listen to famous teachers. The contacts with fellow students, the wide range of active minds is more important to the young student than his reading, or university honors. But of such students few can spend their lives in a university environment. Any one who looks forward to the practice of a liberal profession, for his work in life, must also look forward to a lifetime of study. This is true because we live in an age of science. Medicine, including surgery, and indeed all biological sciences, expand and go forward at a rate never imagined by former generations. In the medical sciences the age of specialization makes extension and progress more rapid than in any other department of human activity. The physician or surgeon who knows nothing of the progress of the last 10 years is behind his time, more behind his time than a whole generation would have been 100 years ago.

The young doctor of medicine who is looking forward to the practice of ophthalmology can find in the larger medical centers many helps to graduate study; in-

ternships, staff positions in the hospitals, local special societies, and graduate courses will all be open to him. But the recent advances in ophthalmology are needed by human beings, in general, wherever they may live. And in spite of the growth of great cities, the majority of people live in country districts and must depend on the village or small-town doctor for medical advice. The smaller cities and towns offer the greatest unoccupied field for the young, ambitious ophthalmologist. The field of practice, as yet entirely unoccupied, offers the best prospect of immediate living, and a professional reputation and development in the place he has selected. The opportunity to apply and profit by what he has learned must always be attractive to the young recruit of the medical profession. For the younger ophthalmologists, the continuation of their studies will depend upon recognizing that continued study is necessary and is still possible without the facilites and opportunities they have elsewhere enjoyed.

The real student is not created by, and is not dependent upon, scholastic institutions and surroundings. If he wishes to learn and keep abreast of others in his special studies he will have to make his own plans and assemble his most important facilities. It is not the existence and assemblage of books that makes reading possible. It is the interest in reading and the determination to keep it up at some cost of effort and even financial outlay. Every practicing ophthalmologist must have some books to which he may turn whenever a patient presents certain characteristics of disease different from anything the doctor has before encountered. Even in a city with the largest libraries, he must have some books in his office, some with which he is already familiar and in which he can most quickly hunt out the new facts and the particular point of view that his patient needs. It is not enough to have the right book, it is essential to be familiar enough with the book to be able to refer to what bears upon the particular case.

The same is true of special journals. They must be looked into, and new facts recorded in each number added to one's mental stock and arranged for future reference. The isolated student may be more dependent on his own little library, and the separate numbers of the journals he takes than the one who remains in a university position.

But one who has fewer books to consult may lose less time in searching for the particular fact or idea that he needs. It may be worth a good deal in money and effort to build up one's own library; but it is an expenditure that the real student will always find profitable.

Abstract truths are better understood, and more certainly retained, when enforced by concerte illustrations. Among general textbooks of ophthalmology, a few of the more recent stand out as preeminently worthy of attention. De Schweinitz's "Diseases of the eye" was issued in 10 editions, extending from 1892 to 1924. Every one of these editions was carefully edited. The author was keenly conscious of what was going on in the ophthalmic world around him, and sought to add to his book everything of value in current literature. The same was true of the "Text-book" of Ernst Fuchs, especially as translated and edited by Alexander Duane. Whatever was new in American or English literature, and of general importance, was submitted by Duane to Professor Fuchs and, receiving his approval, appeared in the American edition. "The eye and its diseases," edited by Conrad Berens, intended by the publishers to replace the book of de Schweinitz, when he ended his editorial career, has characteristics of its own that entitle even its first edition to rank with the others mentioned. Among the 92 contribu-

tors, 39 lived outside of the United States. In this way it represents the recent progress of the world in scientific ophthalmology.

These works illustrate the possibilities for the student of ophthalmology who must depend largely upon his own library. Of course there are special branches, each having literature of its own. The book of Donders on "The accommodation and refraction of the eye," has not been superseded. The works of Vogt, Koby, and Harrison Butler on "Biomicroscopy of the eye," give the isolated student the best literature available to the dweller in great medical centers. The same may be said of the works of Ida Mann on "The embryology of the eye and its developmental abnormalities." The four volumes of Sir John Parsons on "The pathology of the eye" have not been superseded by any more recent publication. "The clinical physiology of the eye," by Adler, is an excellent beginning for the individual study of ophthalmology.

Since the present expansion of ophthalmology is the most imperative reason for continued study, the student, wherever he is, must have access to current journals. Three of these published in English are of important value to all English-speaking ophthalmologists. In addition to this Journal, the Archives of Ophthalmology, started by Herman Knapp in 1869, and the British Journal of Ophthalmology, which, in 1917, replaced the Ophthalmic Hospital Reports, the Ophthalmic Review, and The Ophthalmoscope, each contains enough original new matter, published in English, to enable one to keep in touch with the progress of ophthalmology. Each contains abstracts representing the contents of literature published in other languages and the more important general medical journals of the world.

The real student of ophthalmology will wish access to all its literature, past as well as present. For this, he who lives in a great medical center, with its established libraries, has an advantage. But the isolated student need not despair. Beside the textbooks already referred to, and the files of older medical journals, there are various year-books on medicine and surgery that include references to ophthalmology. In German and French these go back to the beginnings of modern ophthalmology; but in English we have in the Ophthalmic Year-Book, a bridge between the older literature and the abstract departments of the present day. This begins with the literature of 1903, and extends to the abstract department of the present Journal. The advantage of this kind of literature is in the same grouping and arrangement running through successive volumes, making it possible to find anything that has been published on a certain topic. With this the isolated student is at no serious disadvantage, compared with the university teacher. The Year-Book had at one time a yearly circulation of several hundred. Complete sets may be available from the libraries of older ophthalmologists, and are still obtainable at prices very much lower than those for which they were originally issued.

The isolated student who has an interest in his work, and who is forming reasonable personal habits of continued study, need not approach the discussion of current subjects with any fancied complex of inferiority.

Edward Jackson.

GERMANY DISCOVERS THE CROSS CYLINDER

"Peace hath her victories, no less renowned than war."

The so-called internationalism of the medical art is real and substantial. Medi-

cal knowledge is seldom kept as a trade secret, but diffuses freely over the whole civilized world, without such restrictions as are imposed by property rights in many other fields of human activity.

The rate of such diffusion is, however, definitely influenced by certain limiting factors, among which the most important are local and national conservatism, rarity of direct personal contact, and above all the difficulty imposed by lack of a universal language.

Jackson's very practical application of cross cylinders to measurement of the amount of the astigmatic error was first made public in 1887, in a brief paper which is to be found in the Transactions of the American Ophthalmological Society. In 1907 the same author first described the use of cross cylinders for determining the meridian or axis of the astigmatic error. The first detailed and illustrated description of the technique of both cross-cylinder tests was published in 1923.

Until a month or two ago the only other language in which a full description of the cross-cylinder tests had been published was the Spanish. In 1932 the Archivos de Oftalmologia de Buenos Aires issued a translation of a detailed account of the subject which appeared about the same time in the American Journal of Ophthalmology.

Meanwhile the cross-cylinder tests have grown greatly in popularity in the United States and England, although anything like adequate mention of them is still strangely absent from textbooks on ophthalmology in both countries.

Lindner, whose interest in refraction is widely known, and whose lecture tours, as well as his long career as a teacher in Vienna, have made him personally familiar to many American ophthalmologists, now presents (Klinische Monatsblätter für Augenheilkunde, 1939, volume 103, page 273) the first essay on the cross-cylinder tests in the German language. He opens his paper by saying that at the time of his lecture tour in the United States in 1935, Jackson "pressed his cylinder into my hands and recommended me urgently to try it out."

Lindner's article is illustrated by an excellent series of twenty-five photographs and drawings. His enthusiasm as to the convenience and accuracy of the cross-cylinder tests, especially the test for the axis, resembles that of the many American ophthalmologists who have for years made these tests a part of their daily routine.

As to the amount of astigmatism, and as to exact determination of the axis in higher errors, Lindner's experience with the cross cylinder is said to confirm his previous teaching as to the value of cylinder skiascopy. "But in cases of somewhat irregular refraction the cross cylinder showed itself to be very valuable, and superior to velonoskiascopy."

For measuring the strength of the cylinder, velonoskiascopy is in Lindner's opinion unexcelled by any other subjective method when one is dealing with attentive patients. But it demands too much time when one is compelled to guard against inaccurate replies by a patient of slow intelligence. The cross-cylinder test for this purpose, Lindner agrees, is decidedly more rapid. On the other hand, he has satisfied himself that for weak cylinders the correct axis can be discovered most reliably by means of the cross cylinder, so that "for this purpose the cross cylinder has now become quite indispensable" to him.

How many American refractionists, in the course of the past several decades, have expressed themselves in phraseology almost identical with that of our German colleague!

Lindner very properly accords to Jack-

son full credit for use of the cross cylinder by the present-day technique, in which the combination is held in front of the trial frame and quickly "flopped" from one position to the other, with a view to any necessary modification of the strength or axis of a cylinder previously selected. Before Jackson, the principle of the cross cylinder, as elaborated by Stokes* in England in 1849, and as put to practical use by Dennet in the United States in 1886, was applied by inserting a cross cylinder of mechanically variable strength in the trial frame, the strength of cylinder required being determined from the result of raising or lowering the strength of the combination by means of a knob connected with the mounting. The lens element consisted of plus and minus three-diopter cylinders, whose relative positions could be so adjusted as to produce a combination ranging in strength from zero (the two cylinders neutralizing each other) through a series of spherocylindrical effects to a total of six diopters cylinder (the two axes then standing at right angles with each other).

Both in England and in the United States, there are still some ophthalmologists (and even some textbook writers!) whose knowledge of cross cylinders is limited to the test for amount of astigmatism. But the most strikingly original feature of Jackson's development of the cross cylinder is the test for axis, which has become in an important degree "indispensable" to those who have thoroughly familiarized themselves with the method.

The writer of this editorial comment cannot entirely agree with Lindner that use of the cross cylinder for measuring astigmatism "without any other previous examination of the patient" would require a materially greater expenditure of time than is consumed by the ordinary procedures. In the presence of an undilated pupil, and especially for rather low astigmatic errors, skillful combination of the fogging method with the cross-cylinder technique is commonly at least as rapid as any other line of approach.

Some workers have practiced, and an occasional article in the literature has recommended, rotation of the cross cylinder upon its optical center, instead of "flopping" (instantaneous reversal of position) so as to present first one face and then the other of the cross cylinder to the patient's eye. It is therefore of interest to find that Lindner, from his four years of experience with the test, regards the suddenness of the change obtained by the usual procedure as a very important element of the test for either strength or axis.

The following comment by Lindner may be recommended to those American ophthalmologists who have not yet learned the value of the cross-cylinder tests: "Crisp regards application of the cross cylinder to determination of the cylinder axis as more important than its application to determination of the cylinder strength. I am of the same opinion. I believe, however, that many colleagues who have not hitherto made use of cylinder skiascopy, but employ the old-fashioned half-subjective, half objective methods of measuring for glasses, will also value the cross cylinder as an aid in establishing the amount of cylinder. To such colleagues a careful use of Jackson's cross cylinder may serve partly as a substitute for cylinder skiascopy."

In closing his essay, Lindner declares that, while cylinder skiascopy is the best objective method for measuring astigmatism, he now regards the cross cylinder as the most satisfactory aid to subjective control of an astigmatic correction. "I know no other mode of testing

* Spelled "Stockes" throughout Lindner's article.

by which one can determine in a simple way so exactly the correct position of the axis of a weak correcting cylinder as is possible with the help of Jackson's cross cylinder." W. H. Crisp.

(Note: Those who have opportunity to read Lindner's essay will find that the titles of Sections I and II of the description of technique have been transposed, the printer having placed the heading of the strength test above the description for the axis test, and vice versa.)

BOOK NOTICES

TRAITÉ D'OPHTALMOLOGIE. Published under the auspices of the French Society of Ophthalmology. Edited by Bailliart, Coutela, Redslob, Velter, and Onfray. Publishers Masson et Cie, Paris, 1939.

Volume IV (Pathology, continued) comprises chapters on Diseases of the lacrimal apparatus (300 pages) by Valiére-Vialeix of Limoges; Diseases of the conjunctiva (232 pages) by Renard and Nataf of which a large part is devoted to trachoma (by Cuénod and Nataf); Conjunctival diseases in the course of general or adjacent affections (90 pages) by Genet of Lyon; Neoplasms of the conjunctiva (40 pages) by Merigot de Treigny; Diseases of the cornea (150 pages) by Pierre Prélat and Kleefeld; and Diseases of the sclera by Paul Petit.

The volume continues the high standard established by the first two volumes already reviewed (October, 1939, page 1163). It contains a wealth of good illustrations, especially many excellent reproductions of colored photographs and plates. The chapter on the lacrimal apparatus is the most complete of any with which the reviewer is familiar. In addition to a lively discussion of the usual and common conditions met with in practice, it reviews at length rarely encountered affections and disorders, from diagnostic, pathologic, and therapeutic viewpoints.

The chapter on diseases of the conjunctiva is noteworthy for the colored plates of the conjunctiva in the various bacterial infections as seen with the naked eye, the loupe, slitlamp, and when stained with Azur II. It is disappointing to find that the ocular-glandular form of tularemia is not mentioned, even in the discussion of Parinaud's conjunctivitis. This is particularly serious because tularemia has been found to be almost world wide in its extent.

One of the most valuable contributions is that by Cuénod and Nataf on trachoma. They are renowned authorities on this subject. The illustrations and descriptions are superb, particularly those showing the various stages of the follicle and the development of pannus. In the discussion of therapy no mention is made of sulfanilamide, probably because this is too recent an agent in the treatment of trachoma.

Genet disposed of the confusion attending pemphigus of the conjunctiva in a masterly fashion. His description of the various other forms of conjunctival affections are similarly adequate. In contrast to the space devoted to the lacrimal disorders, the pages and illustrations assigned to the diseases of the cornea seem too few in number. Many of the rarer conditions are mentioned too cursorily to be of much value in reference work. Kleefeld's chapter on diverse affections of the cornea is well illustrated with slitlamp pictures in color.

A description of diseases and lesions of the sclera completes the volume.

Volume V (Pathology, continued) begins with a discussion of diseases of the iris by Professors M. Teuliéres and J. Beauvieux of Bordeaux. It embraces the subjects of trauma, iritis, pupil, and tu-

mors, within 78 pages, but the contents although condensed seem adequate for use either as a text or reference. Theoretic and experimental work, however, is not given space, probably justifiably so in a work of this kind. The same authors describe the diseases of the ciliary body and choroid in much the same fashion. The part on tumors of the choroid is particularly good, although the important prognostic work of the U. S. Army Medical Museum has unfortunately been missed. One of the most valuable parts of this portion of the volume, however, is the chapter on tuberculosis of the uveal tract by the aforementioned authors. The subject is covered in all of its phases, both from a theoretic and practical viewpoint, and methods of treatment are thoroughly gone into.

Dr. P. Bailliart covers the subject of diseases of the retina (238 pages) in a masterly fashion, as one would expect. His writing is terse but interesting and the illustrations (in black and white, and colors) are numerous, pleasing, and well chosen. Much attention is directed towards the study of the blood vessels in the various diseases of the retina described. This approach enhances the value and interest of a splendid chapter. His bibliography is generous and modern.

The subject of recurrent retinal hemorrhages in youth has seemed to the editors worthy of a chapter of its own. That their judgment is justified, can be seen at once in glancing through the work of P. Jeandelize and P. L. Drouet. Besides the role of tuberculosis in producing such lesions, the authors devote considerable space to a discussion of the endocrine-gland dyscrasias as etiologic factors and and important therapeutic approach.

Professor Rinsler of Lausanne and Madame Schiff-Wertheimer write a beautifully illustrated chapter of 98 pages on retinal detachment from a diagnostic and pathogenic viewpoint. Therapy is not discussed. Familiar with Gonin's pioneer work, Amsler is a particularly happy choice for such a study.

Dr. J. Mawas, scientific director of the A. de Rothschild Ophthalmic Foundation of Paris, writes on tumors of the retina. His many contributions and skill in classification on this subject are well known. Having devoted much time and study to this topic, he most competently handles the chapter.

Dr. J. Bollack and Madame Delthil write the chapter on diseases of the papilla. When one considers that the subject is limited to the affections of the nerve head alone, the difficulty of writing this chapter can be appreciated. The authors have accomplished their purpose skillfully and well.

Dr. Jean Nordmann of Strasbourg has a short but well-illustrated chapter on tumors of the papilla. The rarity of tumors of the nerve head accounts for the brevity of the chapter.

The final section (116 pages) of the volume is devoted to affections of the crystalline lens. It is written by Professors Duverger and Velter, whose book on "Biomicroscopy of the lens," published in 1930, under the auspices of the French Society of Ophthalmology, is so well known. Most of the magnificent colored plates of this book are used, in addition to many others, both colored and in monochrome. The text is adequately pertinent to the illustrations.

Derrick Vail.

HYDROPHTHALMIA OR CONGENITAL GLAUCOMA. By Dr. J. Ringland Anderson. Clothbound, 377 pages, 116 illustrations. Cambridge University Press, London, 1939. Price 25 shillings.

This monograph is the first comprehensive review of the subject to appear

since 1879 when Gros published in French his résumé of the knowledge of the condition up to that time. Although the incidence of the disease as found among thousands of patients in various eye clinics is relatively very small, the uniformly gloomy outlook for these unfortunate individuals makes this a particularly discouraging and difficult problem.

Dr. Anderson used the questionnaire method to collect case reports of 205 eyes from various parts of the world, and subjects these reports to exhaustive analyses from a great number of standpoints. For this reason, a large proportion of the conclusions reached are on a statistical basis.

Of particular value and interest is a discussion of the comparative anatomy of the filtration system. The structures of the angle in numerous animals, including several Australian types to which the author had access, are described and illustrated. The embryology and anatomy of the human eye are also detailed in this chapter. Etiology, differential diagnosis, pathology, and pathogenesis are discussed at length, with special consideration accorded to the notable association of hydrophthalmia with facial nevi and generalized neurofibromatosis.

Various forms of treatment tried in the past are reviewed, and finally all the different operative methods used by numerous modern surgeons are analyzed as to their results. Miotics are deemed worthy of trial, but in general the only hope seems to be in one of the fistulization operations.

For the busy practitioner in search of specific information, the analytical nature of much of the text might make literal reading somewhat tedious, but the summary at the end of each chapter presents a concise review of conclusions reached by critical analysis of data in the general subject matter of that chapter. The book represents a careful and thorough consideration of available knowledge, is well indexed, and presents an extensive bibliography at the end of each chapter.

George A. Filmer.

CLIO MEDICA: OPHTHALMOLOGY. By Burton Chance, M.D. Volume XX of a series edited by E. B. Krumbhaar, M.D. Clothbound, 257 pages with 6 illustrations. Published by Paul B. Hoeber, New York, 1939, price $2.00.

This book is the twentieth volume of a series of primers on the history of medicine published under the general title of "Clio medica." Each volume aims to present the history of an individual phase of medicine, as the progress of medicine in a certain country, or the story of the development of a particular specialty. The series is uniform in format, the volumes being conveniently small and compact.

In this volume the progress of ophthalmological knowledge has been traced from the time of the earliest written records to the present period. In general, the presentation of the history of eye diseases has been done in a chronological order, but in many instances the development of certain specialized phases of ophthalmology are discussed in detail. There are 26 chapters, some of the headings of which are: Ancient ophthalmology, Discoveries in the sixteenth century, Cataract in the eighteenth century, Sympathetic ophthalmitis and focal disease, The refraction of the ametropic eye and spectacles, The coördination of eye movements, Color vision and color blindness, Ophthalmic surgery, Therapeutic agents, Ophthalmic hospitals and teachers, and Ophthalmology in America.

In spite of its small size, this outline of ophthalmic history is surprisingly complete, very readable, and extremely inter-

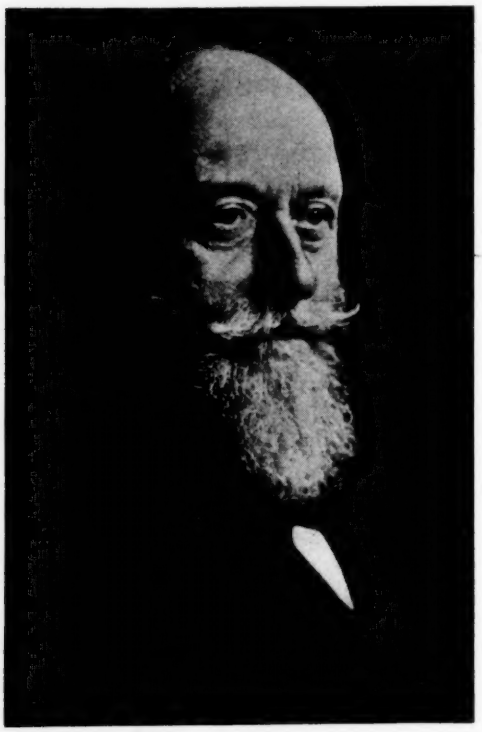
esting. It is unique, filling a definite need, as no work of this type is available in English. This book should prove to be of value, not only to the student of medical history as a source of reliable data and as a readily available reference work, but also to the busy practicing ophthalmologist as a means of gaining pleasantly an excellent concept of the origins and background of his specialty.

T. E. Sanders.

OBITUARIES

PROF. ANTON ELSCHNIG, M.D.
1863-1939

On November 13, 1939, Dr. Anton Elschnig, retired professor of ophthalmology of the German University of Prague, died in Vienna following an automobile accident. He was 76 years old and since his retirement had enjoyed a life dedicated partially to work, partially to rest and sport in the beautiful woods of Marienbad.



Dr. Anton Elschnig

What a contrast to his former life, a restless one, full of work from morning until evening, crowned by the success of a world-renowned name, the grateful veneration of many pupils at home and abroad, and the thankful memory of thousands of patients!

Anton Elschnig was born August 21, 1863, in Graz, South Austria, the son of a high-school teacher. He took his degree from the University of Graz, where he became assistant professor in 1892. With the same rank he entered the famous University of Vienna in 1895. Five years later he was promoted to be associate professor and, after the famous operator Czermak's death in 1907, he was nominated Professor Ordinarius and director of the eye clinic of the German University in Prague. This honorable call was the result of a series of excellent anatomical investigations upon the optic-nerve head. Microscopic investigations were Elschnig's hobby in Graz and in Vienna; he made the slides himself, carefully choosing fixation fluids, exactly manipulating the microtome, and comparing different methods of staining. The results of these studies are decisive today and will remain so in the future. There were many other publications demonstrating Elschnig's interest and ability in clinical questions. In Prague, he became especially active in surgical questions and in studies regarding glaucoma and sympathetic ophthalmia. All this was in addition to indefatigable teaching activity and a private practice increasing from year to year. His special interests were the development of reliable and safe operative methods for extracapsular and intracapsular cataract extractions, keratoplastic, and antiglaucomatous operations.

Working with the genial bacteriologist,

Bail, he explained sympathetic ophthalmia by anaphylaxis. In the years after the World War, he and Axenfeld, became co-editors of the excellent third edition of the great Graefe-Saemisch Handbuch. With the aid of the best collaborators he edited two extensive volumes of "Operationslehre," a standard text for every oculist interested in operative questions. Later on he described the pathology of glaucoma in Lubarsch-Ostertag's Handbuch; collaborated in several editions of Axenfeld's textbook and published an admirable number of clinical observations each year, and numerous articles for the "Zentralblatt." He found time not only to read but to criticize, sometimes to overthrow, every essay written by his assistants, secondary physicians, operation-pupils, and guests from all continents working in his clinics. He was amazingly diligent and, more by his example than advice, stimulated everyone of his collaborators.

His working day started punctually at eight o'clock and finished at some hour late in the night. During his vacations, he always remained in contact with the clinic and discussed in long letters the rough copies of his pupils' drafts. He hunted in the morning and wrote for several hours in the evening, ordered some books to be sent to him to Grundelsee, in the Austrian Alps, asked about an interesting case, or for the results of current investigations. When he returned from vacations he seemed younger and more assiduous than before. During semesters he took holidays for Christmas, Easter, and Whitsuntide. The teacher's vacations were El Dorado for the younger staff for the chance to use the enormous operative material themselves.

No member of the clinic was allowed to operate until he had performed a certain number of operations on pig's eyes and cadavers. Having completed these preparations satisfactorily the young man was proud to see his name on the large cardboard, winking at him in one of the four 2 by 4-meter-wide windows of the extensive and bright operating room. The horizontal top line of this card showed the names of all typical operations and on the margin at the left were the names of the doctors. Every operation was registered by date in the corresponding part. No mistake was possible: the second secondary physician must not do his sixth trephining if the first secondary had only five on the cardboard. Two indispensable qualifications were required to obtain permission to operate; namely, an aseptic conscience and ambidexterity. In this regard Elschnig was merciless. When he noticed unskillful hands, he advised special exercises in drawing and writing with both hands, and none was allowed to operate before he became ambidextrous. Some points located at different distances on a sheet of paper, were to be connected by fine straight pencil lines in a certain tempo with right and left hands. Then, curved lines were drawn and repeatedly followed by the pencil, taking care that the second up to the twentieth line did not emerge from the first pattern line. Pupils unable to do that were advised to leave the clinic and to devote their time to another specialty. Ordinarily, the assistants watched the operative work of the secondary doctors and pupils, but often, especially when an appointive post was in prospect, Elschnig himself watched every step of the applicant's operation.

Rigorous in his demands towards his own work, he was rigorous towards the staff, too, but a true friend to anyone who was able to fulfill his high claims. Operating on 20 to 30 patients a week, he always was surrounded by spectators, and his work was worthy of being watched by everyone. Until he retired in 1934, he constantly tried to improve his work.

He always emphasized that he was learning and insisted it was the duty of any clinic to execute all reasonable operative methods, to compare the results and to adopt the best procedure—*omnia probate, optima tenete*. Therefore a large number of publications from Elschnig's clinic are dedicated to statistics about operative methods, but they are not dry enumerations of results, they are rather comparative investigations, one following the other, complementing, correcting each other.

The patient-cards had to be in exact order. Printed forms gave clear spaces for history and complaints, large enough to accepted completions, pedigrees, descriptions of former operations, and other data. On the first page, there was a space for vision before and after correction and for registering all following operations. On the second page was a ruled and printed scheme giving the general condition first, and then the ocular status printed on separate sheets. After 1918, the records were typewritten, always using the left side of the sheet for the right eye and *vice versa*. Drawings, with and without colors, photographs, and in certain cases moving pictures had to complete the findings. "One good picture explains more about the case than hundreds of words" said Elschnig repeatedly. He liked to draw interesting findings directly into the record. Fields of vision and X-ray films were added, the principle being exactness and clearness. During the years that old "Klinikdiener" Wenzel collected and distributed the patient-cards, there was an admirable arrangement of the cards in spite of an odd phonetic alphabet, chosen by this former artillery man, who had an excellent memory for thousands of interesting cases. Elschnig used to say: "As long as Wenzel is able to find everything, he may have the CH after the K or after the B." Wenzel's task was to bring the specimens from the operating room to our large and well-equipped laboratory on the second floor. He never lost or mixed up one of them in spite of the enormous number of operations and the rush of one following the other. He was also the guardian of the experimental animals living in the basement and treated by him in an extraordinarily able and humane manner. For private operations, Wenzel had to prepare instruments in the sanatoriums and was very proud of being charged with this responsible task. Elschnig used only the best instruments and educated our excellent manufacturer, Frantisek Koutny, to make the best of knives, scissors, forceps, retractors, and so forth. He never allowed an instrument to be used without trying its exactness for all demands.

Elschnig executed his educational duties very conscientiously. Every year from October until December and from January until June, five days a week from 10 to 11 o'clock he gave lectures to the students. The immense material of the Prague clinic allowed him to proceed methodically and to show to the students, cases corresponding to the chapter in discussion. His lectures were really thrilling and always emphasized the relations to general medicine, anatomy, histology, physiology, optics, and chemistry, neurology, rhinology and surgery. As students we found the level of his lectures very high; later on, as his assistant, I was delighted to hear his lectures, to recapitulate and to complete my knowledge, for he always added his newest experiences and the latest reliable findings of the international literature, very highly estimating American publications.

For teaching he used all auxiliary methods and, in spite of his age, sprang youthfully along the three large blackboards of the lecture room. He drew quickly and exactly most complicated topics, explained large illuminated wall pictures, showed lantern slides and mov-

ing pictures, demonstrated patients, and demonstrated physiologic relations on his own eyes or on a student's eyes. Elschnig's gift for drawing was as wonderful as his operating gift.

About 10 students, in rotation from all the two to three hundred enrolled students, had to sit in the auditorium's first row, each group one week, and to inspect the patients before they were demonstrated to the other students. Woe to the poor student who dared to touch an eye without preliminary disinfection of his hands or who would not repeat this ceremony before touching the same patient's fellow eye. Not only in the wards and in the treatment rooms, but in the lecture rooms as well as in the ophthalmoscopic, perimetric, and slitlamp cabinets there were large basins filled with a disinfectant solution, oxycyanide of mercury 1:5000. Every doctor or nurse had to clean his fingers before and after touching a patient's eye. The dressings, in the rooms reserved for patients immediately after operations, were done every day by Elschnig himself, or by his oldest assistants, who for many years before had been allowed to dress patients seven or more days after operation. The oxycyanide basin was pushed by a nurse from easy chair to easy chair (all of iron, like the beds, tables, screens all over the clinic) when Elschnig or the oldest assistant did the dressings. Younger assistants, substituting for the older ones during vacations, had to leap through the large room from the patient's bed or chair to the centrally located basin. Old Anna, queen of the operating rooms since Czermak's time, and the old employee Wenzel, claiming to have served Sattler, Czermak, and Elschnig, would not have touched the basin to hand it to a "young doctor" even if he had been in service three or four years, but not yet first or second assistant.

In examinations, Elschnig was very severe but not equally so to all. To a student exactly describing his findings or drawing a nice fundus picture, he passed over some defects in theoretical questions and figures. However, a student who forgot to disinfect his hands before or after investigation of an eye, or forgot to look first at the patient facing a large north window, or did ophthalmoscopy before transillumination of the media, could expect a bad mark. Ignorance of the technique of oblique illumination was fatal. There were some facts of principal importance, repeated by Elschnig all through the lectures, to be fixed in the student's memory for all his life. These were relations of ophthalmology to general medicine, aseptic conscience, diagnosis of trachoma, attention to dangerous corneal diseases, care never to damage an operated or injured eye by awkward manipulation of the lids, always to think of glaucoma (named by Elschnig in a figure of speech taken from Spanish bull fights: "ruddy cloth of the oculist"), never to blame cataract for loss of vision before excluding other causes hidden by incipient lens opacities, and a lot of other things, some of which seem bagatelles, but taken together give an extremely good training to undergraduates.

Always busy, and in spite of an enormous private practice, Elschnig found time to discuss with his assistants cases, scientific problems, and questions of private life. He ordered staff meetings every Wednesday. There, interesting cases and the actual findings of our scientific investigations were discussed, topics for further investigations ordered, good results designed to be presented two days later, on Friday, to the Medical Association, the best results reserved for the meetings of the Ophthalmological Society or for the the meetings in Heidelberg.

Before 1918, all doctors had to be present in the clinic every day including Sundays and holidays; only Sunday and holiday afternoons were free. After 1918,

every second Sunday was free, the other Sunday we had to be busy until noon and on call in the hospital from noon until Monday morning. After finishing private dressings on Sundays, Elschnig liked to see the clinic for one or two hours and to have a chat with the doctors on duty. On these Sundays, having more time, he often showed to us his extremely fine microscopic preparations, done by himself many years earlier or dedicated by colleagues all over the world. When in especially good humor, he recounted anecdotes of his Vienna chief and teacher, Schnabel, sometimes imitating his voice. Having an excellent classic education, he sometimes loved to use half Latin phrases; for example, "minima non curat praetor," when he liked to turn boring administrative work over to his assistants, or "fortiter in re, suaviter in modo," when there were differences between our clinic and another. On these Sundays, too, he discussed the future development of his pupils, knowing everyone's abilities, prospects, and familiar conditions. Knowing the situation in the small towns of our republic, he was able to recommend everyone to open his practice here or there; he was delighted to give good recommendations to local hospitals and authorities, to give advice concerning private life, including sometimes questions of marriage.

He had a rare capability of judging personalities from the first glance and did not like to change his view until compelled to do so by facts. In society, he was a charming conversationalist, having good taste in matters of art and interest in social and medical coöperative questions. He really had a sort of artistic vein, a good voice, and liked music, detested smoking, liked sweets and wine of which he was a connoisseur too. As a student in Graz, he was a member of a fraternity cultivating the old students' customs and singing; later on he became a member of the world-wide society of Schlaraffia, cultivating "art, good humor, and friendship." Nearly every Saturday he forgot the reality of this world for some hours sitting in the beloved "Burg" between old-fashioned pictures, sculptures, and standards; and wearing, as all "knights" of this "order," a fool's cap in black, yellow, red, and blue, ornamented with small bells and other symbols of merriment. Here he liked to hear musical productions, played by members of our theaters and orchestras, sometimes themselves composers, and recitations of poems, written for this ephemeral situation or recited from our best poets' works; here he saw pictures and sculptures just finished by painters and sculptors, members of the Schlaraffia.

In his home, too, Elschnig loved to see friends, to be surrounded by good books, good pictures, and good music. His wife, a typical Austrian beauty with noble traits, was the most charming lady, unaffected and estimable, speaking a wonderful, soft Viennese dialect. The best housewife and mother, she supervised the home as well as the office, in the latter invisible to patients and visitors, but knowing how to clean and preserve all instruments and able to decipher the patients' records, written by Elschnig personally in his frizzled abbreviated script. In his private life Elschnig was economical, he never owned a car and preferred to use street cars rather than cabs. His beard was never cut and reached about six inches below his chin: he used to say humorously that he did so for saving time and money, but actually it was an old tradition of the physicians before 1900 and you may see similar venerable beards in old pictures of physicians and surgeons, not only from Austria.

Elschnig was a member (among other important scientific societies) of the Society for Art and Science in Prague, a member of the Board of the Ophthalmo-

logical Society of Heidelberg, and president of the German Ophthalmological Society of Heidelberg, in Czechoslovakia.

Elschnig's oldest daughter is married to a physician of Marienbad. His son Hermann was an assistant in his clinic for many years, became an excellent operator, too, and opened his practice in Zniam in Moravia. His youngest daughter is a singer in Vienna.

For all of us who were so fortunate as to be his pupils, Elschnig will be unforgettable for his excellent diagnostic, operative, scientific, and teaching qualities. He concentrated in his comprehensive memory the highly developed modern ophthalmology, a great part of which is indebted to him for the high plane of evolution it has reached.

Karl W. Ascher.*

HARVEY CUSHING

1869-1939

This great neuro-surgeon was born in Cleveland, Ohio, on April 8, 1869. He deserves notice here for the contributions he has made to the neurology of vision, and the physiology and pathology of the pituitary body, with its effects on the field of vision. He was graduated at Yale and then went to the Harvard Medical School, where he took his M.D. in 1895. He served as house officer in the Massachusetts General Hospital. From there he went to Johns Hopkins Hospital to work under Professor Halstead, to learn his accurate methods and meticulous care to avoid injury to any tissue that was to be left in the body. In spite of Dr. Halstead's objection, "Why Dr. Cushing, we had only two cases of brain tumor last year," he chose neuro-surgery for his work, and went to Europe to spend two years on experimental neurology with Sherrington, Kocher, and others.

Returning to Johns Hopkins be became the neuro-surgeon of the staff and developed a close friendship with Osler, which furnished the foundation for his "Biography of Sir William Osler," the greatest medical biography of our time. In 1910 Cushing published his book on the "Pituitary body and its disorders. Clinical states produced by disorders of the hypophysis cerebri." The close relations between this organ and the visual tracts made the book of great interest to ophthalmologists, and it threw new light upon impairments of the visual fields. In 1910 he was made Professor of Surgery at Harvard University, and Surgeon-in-Chief to the Peter Bent Brigham Hospital.

In the World War he became the senior consultant in neuro-surgery of the American Expeditionary Force; was honored by the award of a Distinguished Service Medal from the United States, the Order of the Companion of the Bath from Great Britain, and the position of "Officer of the Legion of Honor" from France.

Returning to Boston his work in neurology and neuro-surgery gained world-wide recognition. In 1929, a special number of the Archives of Surgery, emphasizing his qualities as a teacher, contained 82 papers contributed by men holding important medical and surgical positions both in America and Europe. In 1932, at a date previously fixed by himself, he retired from practice, and the next year was made Sterling Professor of Neurology in Yale University, a position he held until 1937. But his literary activity continued, and on his seventieth birthday a bibliography of his writings was issued, including 305 addresses, papers, and reports, many containing items of important historical interest. His successor at Harvard, Professor Elliott C. Cutler, has said: "He himself would like most to be re-

* Formerly Professor Extraordinarius in Ophthalmology of the German University of Prague.

membered as one who upheld the first tradition of our profession—Let nothing be neglected that can benefit a patient." To the end of his life he kept in close touch with colleagues who could report from time to time on the continued progress of his important operative cases.

Edward Jackson.

CORRESPONDENCE

Presentation of Portrait of Dr. Edward Jackson at Wills Eye Hospital

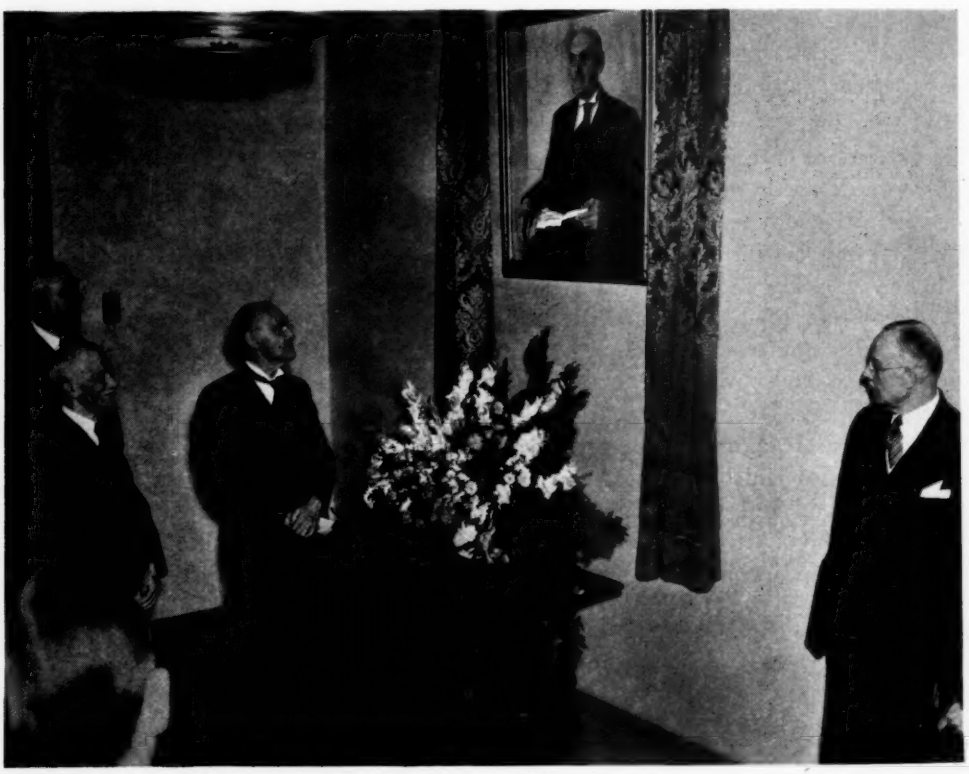
Editor,
American Journal of Ophthalmology

On Thursday, October 19, 1939, a portrait of Dr. Edward Jackson was presented to the Wills Eye Hospital in Philadelphia with appropriate ceremonies. Dr. Jackson's early professional life was spent in Philadelphia, where he was one of the surgeons to the Wills Eye Hospital at the time of his removal to Denver.

The portrait was arranged for by subscriptions among Dr. Jackson's many friends, and was presented by Dr. William Zentmayer in behalf of the donors, and accepted for the Hospital by Mr. Ernest Trigg, the Chairman of the Board of City Trusts, which has charge of the Hospital. There were present many of Dr. Jackson's friends, who were attending the Session of the American College of Surgeons, and also others who were interested in the Hospital.

Our photograph of the occasion shows the portrait of Dr. Jackson, Dr. Jackson, Dr. Zentmayer, Mr. Trigg, and Mr. Stephen Wierzbicki, Superintendent of the Hospital.

(Signed) E. C. Ellett.



ABSTRACT DEPARTMENT

EDITED BY DR. WILLIAM H. CRISP
ASSISTED BY DR. GEORGE A. FILMER

Abstracts are classified under the divisions listed below, which broadly correspond to those formerly used in the Ophthalmic Year Book. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is only mentioned in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. General methods of diagnosis
2. Therapeutics and operations
3. Physiologic optics, refraction, and color vision
4. Ocular movements
5. Conjunctiva
6. Cornea and sclera
7. Uveal tract, sympathetic disease, and aqueous humor
8. Glaucoma and ocular tension
9. Crystalline lens
10. Retina and vitreous
11. Optic nerve and toxic amblyopias
12. Visual tracts and centers
13. Eyeball and orbit
14. Eyelids and lacrimal apparatus
15. Tumors
16. Injuries
17. Systemic diseases and parasites
18. Hygiene, sociology, education, and history
19. Anatomy, embryology, and comparative ophthalmology

1

GENERAL METHODS OF DIAGNOSIS

Alaerts. **A pupillometer for direct reading.** Bull. Soc. Belge d'Opht., 1939, no. 78, p. 86.

This instrument magnifies and has a micrometer and a constant source of light.

Sampaio, M., and Rezende, Cyro de. **How to diagnose ocular tuberculosis.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 2, pp. 605-613.

The author discusses at length the different means at the disposal of the ophthalmologist for diagnosis of ocular tuberculosis: eye symptoms, biopsies, tuberculin reactions, inoculations of laboratory animals, index of Velez, and hemosedimentation. Differential diagnosis is also discussed. The necessity of close collaboration between the internist and the ophthalmologist is emphasized by the author.

Ramon Castroviejo.

Sauberman, G. B. C. **The influence of intensity of illumination on the measurement of size of the blind spot in the normal eye.** Ophthalmologica, 1939, v. 97, Aug., p. 364.

There is great variation in the reported size of the blind spot. The author tabulates 25 such reports gathered from the literature to support this statement. He points out 13 details in apparatus and method that may be responsible for this variation. He systematically investigated the influence of light intensity and found that in a brilliantly lighted room the excitation in the entrance zone of the optic nerve is extinguished by contrast. Hence, the blind spot is larger when measured in bright light than in a relatively dark-adapted eye in artificial illumination. F. Herbert Haessler.

Schepens, Ch. **Concerning perimetry.** Bull. Soc. Belge d'Opht., 1939, no. 78, p. 73.

The purpose of the writer is to describe a simple method of exploration of the visual fields, based upon his observations conducted in the London clinics, Moorfields in particular. He

uses a black background for white test objects and a gray background for colored test objects, employing the smallest test object visible in the region being explored. It is noted, for example, that a white stimulus which subtends an angle of 0.25 degree at 330 mm. gives a larger field than a stimulus which subtends the same angle at 2 meters. This is obviously due to the brighter illumination of the nearer stimulus. The writer uses the Bjerrum screen at 2 meters for examination of the middle field of vision, but prefers colored stimuli with reduced illumination. He notes that the sensitivity of the retina for white is greatest in a condition of dark adaptation, and for colors in a condition of light adaptation and in the macular region. In cases in which a lesion is easily determined by the use of white test objects it is seldom necessary to employ colored objects. The best results with white fixation objects are attained by using a 3-mm. white disc with a black center, thus aiding the eye in maintaining steady fixation. In using the Bjerrum screen the head of the patient should be firmly fixed. The blind spot is delimited at 2 meters with a white stimulus of 30-mm. diameter. The 2/2000 and 1/2000 white isopters are then examined, and finally the central zone, with 10-mm. and 15-mm. colored test objects. It is advisable for the examiner always to use the same instruments and under the same conditions. The personal factor is of great importance and not all examiners using the same methods obtain equally good results. (3 plates, 10 references.)

Jerome B. Thomas.

Thomson, A. M., and others. **A study of diet in relation to health. Dark adaptation as an index of adequate vitamin-A intake. 3. Relation of diet to rate and extent of dark adaptation.** Brit. Jour. Ophth., 1939, v. 23, Nov. pp. 697-723. (See Section 10, Retina and vitreous.)

Weve, H., and Ziedses des Plantes, B. C. **Planigraphic demonstration of a sinus shadow with orbital complication.** Ophthalmologica, 1939, v. 97, Aug., p. 346.

In the planigraphic method of roentgenography, the plate and the X-ray tube are moved circularly in opposite directions in such a manner that a straight line passing through them will describe a pair of cones of rotation whose apices lie at a point in that part of the tissue which is to give a clear image on the plate. This method gives strikingly clear pictures of a small area. Illustrations show how films made by the planigraphic method demonstrated the presence of sinusitis with secondary optic neuritis and made it possible to give a correct diagnosis when other methods of clinical examination had failed completely.

F. Herbert Haessler.

2

THERAPEUTICS AND OPERATIONS

Bouton, S. M., Jr. **Vitamin C and the aging eye.** Arch. Intern. Med., 1939, v. 63, May, p. 930.

About 150 persons (officers, employees, and patients) at the Hastings State Hospital in Nebraska were studied experimentally and clinically. After a review of the literature and a description of the experiments, the author concludes that ascorbic-acid deficiency can be held at least partially responsible for impairment of vision associated with senescence of the human eye, and that the administration of ascorbic acid by mouth in adequate doses can counteract this process. The

senile cataractous lens, however, although showing a definitely subnormal ascorbic-acid content, is apparently not affected by the administration of ascorbic acid by mouth, even in very large amounts. Little is to be gained by continuing the administration of large amounts of ascorbic acid if there is no measurable improvement after daily treatment for two weeks. Use of several vitamins may prove successful when ascorbic acid alone ceases to be of benefit. (29 references, 6 tables.)
Ralph W. Danielson.

Bronfenbrenner, J., and Sulkin, S. E. **Bacteriophage therapy. 2. Prophylactic and therapeutic effect of bacteriophage and of antivirus in experimental infections of the eye.** Jour. Infec. Dis., 1939, v. 65, July-Aug., pp. 58-63.

The authors report a series of experiments testing the effect of bacteriophage on staphylococcic lesions in the eyes of rabbits and guinea pigs. They conclude that instillations of bacteriophage into the conjunctival sac are not sufficient to protect the cornea against severe infection, and that direct application is of no value in treatment of eyes experimentally infected with the staphylococcus. T. E. Sanders.

Bronfenbrenner, J., and Sulkin, S. E. **Prophylactic and therapeutic effect of bacteriophage and of antivirus in experimental staphylococcus infection of the eye.** Amer. Jour. Ophth., 1939, v. 22, Dec., pp. 1321-1325.

Busacca, A. **Ultraviolet rays in ophthalmology with "Infrazon" apparatus and short wave.** Rassegna Ital. d'Ottal., 1939, v. 8, May-June, p. 379.

Busacca employed an ultraviolet apparatus called "Infrazon" in various external diseases of the eye. He obtained good results in trachoma with pannus, in supraorbital and infraorbital neuralgia from various causes, in atonic corneal ulcers implanted upon leucomata, and in epiphora of unrecognized causation. No benefit was found in eczema of the lids or blepharitis. The electrode must be applied to the external surface of the lid and not to the conjunctiva. The series of treatments must be given without interruption.
Eugene M. Blake.

Foster, J. **A bottle and dropper for oily eserine.** Brit. Jour. Ophth., 1939, v. 23, Oct., p. 679.

This bottle has a stainless-steel stopper with an overhanging rim and an attached rod which remains immersed in the solution. It is intended for semi-darkroom purposes. D. F. Harbridge.

Gerasimenko, T. H. **Clinical and laboratory evaluation of diocaine.** Viestnik Opht., 1939, v. 15, pt. 1, p. 41.

The conclusion is that 0.1 to 0.25 percent diocaine instilled into the conjunctival sac produces an anesthesia sufficiently deep and lasting for intraocular procedures. Ray K. Daily.

Meoni, Mario. **Chemotherapy of sulphanilamide in ocular affections.** Arch. di Ottal., 1939, May-June, v. 46, p. 183.

The use of sulphanilamide in ophthalmology is reviewed by the author and the effect of sulphanilamide in experiments carried out on rabbits is described. Following an Elliot trephining, rabbits were inoculated with (1) staphylococcus aureus, (2) staphylococcus albus, (3) staphylococcus citreus, and (4) bacillus pneumoniae of Friedländer. The eyes reacted to the inoculations and prontosil was instilled in the infected eyes. The chemotherapeutic action of sulphanilamide used by local application was found to be very successful in aiding resolution of the in-

fective processes. Intraocular infections were also produced in rabbits' eyes, and prontosil proved to be very efficacious in controlling the infection. The author cites eight clinical cases in which prontosil was used and concludes that the drug was undoubtedly of great benefit in the various ocular infections, but he believes that the usual local therapy should be carried out in addition to the sulphanilamide therapy.

H. D. Scarney.

Mieses-Reif, Maria. **Some clinical experiences with vitamin C in ocular diseases.** Acta Ophth. Orientalia, 1939, v. 1, April, p. 170.

In 80 cases of various eye diseases the urine was examined for vitamin C by a simplified method of Tillman. In half the cases there was no vitamin C present in the urine. The course of these cases was more severe than that of those which were saturated with vitamin C. In certain forms of cataract vitamin C seemed to have an undeniable therapeutic value. Improvement was observed in one case of subcapsular cataract and in five cases of incipient cataract. R. Grunfeld.

Mueller, Friedrick. **Remarks about prophylaxis and treatment of eye diseases with special reference to tuberculosis.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 2, pp. 615-620.

The condition of the entire organism should be considered in treatment of an ocular affection. The importance of tuberculosis as an etiologic factor in ocular diseases is emphasized.

Ramon Castroviejo.

Pinkhof, J. **The content of sulphanilamido-pyridine in the ocular fluids of rabbits after administration of the drug.** Ophthalmologica, 1939, v. 97, Aug., p. 356.

After oral administration of 0.1 gram per kilogram of rabbit, the concentration of sulphanilamido-pyridine in the aqueous rose to from 50 to 100 percent of that of the blood, and in the vitreous, to between 25 and 33 percent. Subconjunctival injections of the blood did not increase these concentrations and local application of the drug in the conjunctival sac did not result in intraocular absorption.

F. Herbert Haessler.

Promtov, V. A. **New anesthetics.** Viestnik Opht., 1939, v. 15, pt. 1, p. 33.

A report on the clinical tests of a series of anesthetic substances designed to replace cocaine. The entire series was found to be unacceptable and inferior to tiocaine, diocaine, or naftocaine, all of which are satisfactory substitutes for cocaine.

Ray K. Daily.

Rezende, Cyro de. **Treatment of ocular tuberculosis according to present-day knowledge.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 2, pp. 635-641.

The author reviews the present status of the treatment of ocular tuberculosis. General treatment, tuberculin therapy, ultraviolet rays, gold therapy and chemotherapy, X rays, radium, Friedmann vaccine, and autohemoimmunotherapy are discussed.

Ramon Castroviejo.

Vakker, A. G. **The use of chloricide in serpiginous ulcers and other ocular diseases.** Viestnik Opht., 1939, v. 15, pt. 1, p. 68. (See Section 6, Cornea and sclera.)

3

PHYSIOLOGIC OPTICS, REFRACTION, AND COLOR VISION

Berens, C., and Stein, L. **Group color-vision tests.** Jour. Amer. Med. Assoc., 1939, v. 113, Oct. 21, p. 1563.

The authors report a method for testing large groups of subjects for disturbances of color vision by employing Kodachrome film to make colored copies of the Ishihara or Stilling plates. Projection of the slides yields good images readily perceived in large audience halls and thoroughly satisfactory for testing the color vision of large groups of subjects. The authors suggest the possibility of educating color-blind persons to differentiate colors by demonstrating color scenes and charts.

George H. Stine.

Blankstein, S. S., and Fowler, M. J. **Visual-acuity tests.** Amer. Jour. Ophth., 1939, v. 22, Dec., pp. 1377-1382.

Boeck, J. **Experiments in correcting corneal astigmatism with electro-coagulation.** Wiener klin. Woch., 1939, v. 52, Oct. 27, pp. 971-974. (See Section 6, Cornea and sclera.)

Cordes, F. C., and Harrington, D. O. **Asthenopia due to vitamin-A deficiency.** Amer. Jour. Ophth., 1939, v. 22, Dec., pp. 1343-1354.

Crozier, W. J., and Holway, A. H. **Theory and measurement of visual mechanisms. 3. ΔI as a function of area, intensity, and wave length, for monocular and binocular stimulation.** Jour. Gen. Physiology, 1939, v. 23, Sept. 20, pp. 101-141.

This highly technical article does not lend itself to abstracting.

Dashevsky, A. I., and Byshmich, D. G. **Transposition of the visual angle from the visual line on the optic axis of the eyeball.** Viestnik Opht., 1939, v. 15, pt. 1, p. 82.

This phraseology the author applies to the calculation of the size of the angle alpha modified by the presence of the angle kappa. He presents a series of tables to take the place of complicated mathematical calculations. He believes that this procedure will prove useful in localization of foci in the fundus.

Ray K. Daily.

Fritz, A. **Contact glasses of organic substance and of individual mold.** Bull. Soc. Belge d'Opht., 1939, no. 78, p. 71.

A brief description of the taking of a cast of the eyeball with dental wax, from which cast a contact lens of "organic glass" is made. J. B. Thomas.

Gailey, W. W. **Transient myopia from sulphanilamide.** Amer. Jour. Ophth., 1939, v. 22, Dec., pp. 1399-1400.

Herzau. **Retrospect and prospect of aniseikonia research.** Deut. Ophth. Gesell., 1938; in Klin. M. f. Augenh., 1938, v. 101, July, p. 97.

Herzau reviews the aniseikonia question, referring particularly to the work of Ames and his associates, and describing their apparatus and methods. Related questions requiring further elucidation are discussed and the author reports certain personal observations.

Percy Fridenberg.

Knapp, A. A. **Vitamin-D complex in progressive myopia.** Amer. Jour. Ophth., 1939, v. 22, Dec., pp. 1329-1337.

Kravkov, C. V. **The effect of the intensity of the auditory stimulus on the color sensitivity of the eye.** Viestnik Opht., 1939, v. 15, pt. 1, p. 100.

The conclusions of this study are

that under the influence of an indirect sound stimulus, color sensitivity is increased for green and diminished for orange. This qualitative response remains unchanged with an increase in intensity of the sound stimulus; the quantitative response increases with an increase in the intensity of the sound stimulus. To the same stimulus different persons may present a different quantitative response in the positive or negative phase. The author attributes the different effect of the same indirect sound stimulus on the green and orange color sensitivity to a difference in the properties of the green and red perception apparatus. Ray K. Daily.

Kreiker, A. **Stabilization, a completion of Steiger's teaching.** Graefe's Arch., 1939, v. 140, pt. 2, pp. 269-277.

The author discusses and enlarges upon Steiger's teachings concerning the congenital or developmental cause of refractive errors. He uses the term "stabilization" in dealing with axial growth of the eyeball in its effect upon the production of axial ametropia.

Charles A. Perera.

Lancaster, W. B. **Report of the American Committee on Optics and Visual Physiology.** Jour. Amer. Med. Assoc., 1939, v. 113, Oct. 7, p. 1413.

This is a condensed report of the various subcommittees.

Lindner, K. **The subjective determination of astigmatism with the Jackson cross cylinder.** Klin. M. f. Augenh., 1939, v. 103, Sept., p. 273.

Lindner describes the Jackson cross cylinder and considers it the best aid for the subjective control of astigmatic correction. C. Zimmermann.

Lokshin, C. I., and Sherman, L. M. **Hörner's syndrome and accommodation.** Viestnik Opht., 1939, v. 15, pt. 1, p. 60.

The objective of this experimental investigation was the determination of the role of the sympathetic in accommodation. In twenty patients an experimental Hörner syndrome was produced by novocaine blocking of the cervical sympathetic. Miosis and ptosis appeared almost immediately after the injection. The state of accommodation was carefully measured before and after injection. In all cases the accommodative function remained undisturbed. Ray K. Daily.

Motzkin, Theodor. **Some remarks on Weinberg's article "The form and extent of the region of indistinct vision in quick forward motion"** (see below). Acta Ophth. Orientalia, 1939, v. 1, July, p. 200.

The author proved graphically the circular form of the space of indistinctness. The diameter of this circle is one meter for a velocity of one km. an hour, a distance covered within three to four seconds. R. Grunfeld.

Smukler, M. E. **Mirror writing in school children.** Pennsylvania Med. Jour., 1939, v. 43, Oct., p. 21.

On the basis of psychometric and ocular examinations Smukler concludes that mirror writing in young children is only a temporary phenomenon lasting from two to twelve months, providing there is no cerebral pathology. He states that social, economic, and physical conditions are the important factors in producing mirror writing.

Theodore M. Shapira.

Thompson, W. M., and Nugent, O. B. **Progressive myopia.** Illinois Med. Jour., 1939, v. 75, March, p. 231.

Statistics on the incidence, predispos-

ing and exciting causes, differential diagnosis, and mechanics of the disease are given. The method of ascertaining the amount of prism base in is described. The prism is added to from 75 to 80 percent of the total spheric correction found. The authors found this management very successful in all cases of axial progressive myopia, less so in the more complicated types.

F. M. Crage.

Weinberg, Erich. **The form and extent of the region of indistinct vision in quick forward motion.** Acta Ophth. Orientalia, 1939, v. 1, July, p. 191.

The author established experimentally the velocity of distinct vision, both central and peripheral, with and without spying eye movements. He determined by calculation the region in space in which a quickly moving observer cannot recognize details, and found this region of indistinct vision to be a torus. The circle producing it had a radius equal to the velocity of the eye divided by the limit velocity. This formula is important with regard to motor-driving, aviation, and the comparative physiology of the senses in animals. R. Grunfeld.

4

OCULAR MOVEMENTS

Coppez, Leon. **The operative treatment of strabismus.** Bull. Soc. Belge d'Opht., 1939, no. 78, p. 27.

To gain moderate effects, either tenotomy or advancement may be recommended, but it is important in convergent strabismus to measure the convergence power, for only in cases in which the latter is excessive should one be content with a tenotomy of the internal rectus. If the convergence is normal or weak, advancement of the external rectus is the operation of choice. Complete tenotomy should be avoided in all cases. In divergent strabismus of the adult, advancement and tenotomy may be performed at the same seance and overcorrection need not be feared. In convergent strabismus the writer postpones consideration of operation until the end of puberty, at which period he notes in certain cases an amelioration of the strabismus. Besides, when the strabismic eye is completely amblyopic (without macular vision), there is no chance of improving the vision even after correction of the deviation. If one is a partisan of the theory of amblyopia from disuse, it is logical to advise operation as early as possible in order to begin reëducation of binocular vision. Nevertheless, stereoscopic exercise is extremely difficult in young children and should be reserved for adolescents. The technique of advancement practised by the writer is that of Van Lint (Ophth. Year Book, 1921, v. 17, p. 57). The operation is in reality a shortening of the muscle, effected on two planes and giving great security. The results are excellent. (5 drawings.) Jerome B. Thomas.

Harms. **Pathogenesis of monocular amblyopia.** Deut. Ophth. Gesell., 1938; in Klin. M. f. Augenh., 1938, v. 101, July, p. 99.

In strabismus, processes of suppression in the interest of undisturbed vision become active in the retina. It was shown that central disturbances necessarily follow and this led to the assumption of a pre-formed inhibitory mechanism. The impulses for this reaction take place in supracortical sensory centers for associative combination, are carried by way of the visual paths in descending nerve fibers to the periphery, and become active in the ganglion cells of the retina. Con-

tinuous central suppression of vision of one eye of an infant results in amblyopia. The suppression takes place whenever a considerable difference in the impressions registered in corresponding retinal points prevents their uniting to form a single one. Such discrepancy may be due to optical errors of one or both eyes or to motor anomalies. The amblyopia of anisometropes is a suppression amblyopia. This is demonstrated in the individual case by the presence of a central scotoma and a lowering of the pupillomotor index. As improvement is possible, the practical conclusion is that every case of monocular amblyopia should be subjected to orthoptic exercises.

Percy Fridenberg.

Hoorens. **Concomitant strabismus.** Bull. Soc. Belge d'Opht., 1939, no. 78, p. 10.

As the first speaker in a symposium on concomitant strabismus, Hoorens takes up the following subjects: examination of the patient, indications for orthoptic treatment, and how to conduct such treatment. If hyperopia is present he prescribes a full correction and he asserts that such correction suffices to cure most cases of periodic convergent strabismus. In divergent strabismus with a moderate degree of myopia he orders a slight overcorrection, followed by stereoscopic exercises. He is convinced that most, if not all, cases of amblyopia are due to disuse and that they are curable. In children too young to wear glasses one should cover the good eye for several weeks and then alternate the cover. The use of atropine in the good eye is disapproved because in spite of the instillations the little patient continues to squint and to use the good eye. The speaker describes his method of using the stereoscope, illustrates the text with seven figures, and presents three case reports. In reply to the criticism that the orthoptic treatment of strabismus demands an inordinate amount of time and patience, he states that in the great majority of cases the treatment is simple. Careful refraction and, if amblyopia is present, covering the good eye, should require three or four weeks, including three or four consultations. To make sure that the strabismic eye retains its visual acuity, the patient should be seen two or three times a year. When the eye straightens spontaneously, a few stereoscopic exercises should be ordered, at most requiring four or five consultations.

Jerome B. Thomas.

Kleefeld, G. **Pleophotography as a record of strabismus.** Bull. Soc. Belge d'Opht., 1939, no. 78, p. 60.

One may profit by using an industrial process which consists in taking photographs of a person in a number of positions, even as many as 48 different views. The process is inexpensive. The photographic record is peculiarly valuable, and possesses the advantage in medicolegal cases of being a commercialized process carried out by nonmedical persons, whose testimony thus has an independent value.

Jerome B. Thomas.

Malbran, J. and Adrogué, E. **Concerning monocular diplopia in strabismus.** Arch. de Oft. de Buenos Aires, 1938, v. 13, Dec., p. 684.

This is in the nature of a reply to an article by Sverdlick (Amer. Jour. Ophth., 1939, v. 22, p. 1176). Several controversial aspects of monocular diplopia are discussed.

Edward P. Burch.

Moers. **Tenotomy with forced abduction.** Bull. Soc. Belge d'Opht., 1939, no. 78, p. 58.

A short report illustrated by photographs.

Rehsteiner, K. **Contribution to the therapy of squint amblyopia.** Schweizer med. Woch., 1939, v. 69, no. 43, p. 1036.

The author presents seven cases of amblyopia secondary to convergence. The earliest age at which treatment (covering of the better-seeing eye) was started was three years, and the latest, five years. Covering was intermittent, lasting from one hour to the entire day over a period of five months. In all cases vision was markedly improved.

Theodore M. Shapira.

Sergeev, I. V. **Determination of the angle of strabismus.** Viestnik Opht., 1939, v. 15, pt. 1, p. 86.

The author facilitated the determination of the angle of strabismus on the perimeter by attaching a fixed light to the center of the perimeter and a movable light on the arm. Ray K. Daily.

Vogt, Alfred. **A new method of correcting paralytic strabismus in contracture of the antagonist.** Klin. M. f. Augenh., 1939, v. 103, Sept., p. 296.

After thorough detachment of the tendon of the contractured internus muscle, doubly-armed sutures are placed around the tendons of the intact superior and inferior recti, then carried temporally through the skin at the lateral edges of the respective lids and tied over gauze pellets. (Photographs.) C. Zimmermann.

Weekers, L. **Two operations for strabismus.** Bull. Soc. Belge d'Opht., 1939, no. 78, p. 43.

One operation practiced by the writer for 15 years has to do with muscle advancement in cases of extreme divergent strabismus, both concomitant and paralytic. The internal rectus muscle which is to be advanced is not sutured to the sclerotic but is dissected from the globe, drawn over the cornea, and attached to the area covering the tendon of the external rectus. The cornea is thus entirely covered by a flap of conjunctiva and muscle tissue. After about a week the stitches are removed and the tongue of conjunctiva and muscle covering the cornea is trimmed. By this method a single advancement may effect a shortening of 35 or 40°, and the ocular movements are not limited. The second operative procedure described by the writer is a shortening of the rectus muscle and is applicable to both convergent and divergent strabismus. The advantages and disadvantages of advancement and of shortening are discussed at length by the author, who concludes in favor of shortening. (7 figures.)

Jerome B. Thomas.

5

CONJUNCTIVA

Abdulaev, G. G. **Conjunctivoplasty.** Viestnik Opht., 1939, v. 15, pt. 1, p. 91.

The author uses mucous membrane from the lip. After freeing the orbit from contractures, he covers the raw areas with mucous membrane, which is sutured in place and held there by a sterile prothesis. He believes that mucous membrane is the most suitable material because it takes rapidly, easily, and firmly; does not shrink or macerate; and it simulates the conjunctiva in appearance. Ray K. Daily.

Allen, J. H. **Experimental production of conjunctivitis with staphylococci.** Amer. Jour. Ophth., 1939, v. 22, Nov., pp. 1218-1226.

Amorim, Theonilo. **A new technique of diathermal coagulation for the treatment of pterygium.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 2, pp. 832-833.

A new method of electrocoagulation for pterygium is described. The passive electrode has the form of a ring which completely surrounds the pterygium. The active electrode is used with a current of 100 milliamperes to electrocoagulate the apex of the growth. The author claims that with this treatment the recurrence of pterygium is less frequent than with any other surgical method. Ramon Castroviejo.

Anderson, W. B., and Byrnes, T. H. **A case of rhinosporidium of the conjunctiva.** Amer. Jour. Ophth., 1939, v. 22, Dec., pp. 1383-1388.

Aprigliano, Orlando. **Intravenous treatment of trachoma with Cusylol.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 2, pp. 731-734.

The author advocates intravenous injections of Cusylol (citrate of copper) in the treatment of trachoma.

Ramon Castroviejo.

Assis Brasil, E. de. **The value of the index of Velez in the etiology of phlyctenular affections.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 2, pp. 627-634.

(According to Velez in tuberculous conditions the number of polymorphonuclear leucocytes of two nuclei is greater than of those of three or four nuclei. This phenomenon is called by Velez "nuclear inversion.")

The author, after studying the index of Velez in phlyctenular affections, arrives at the following conclusions: (1) The index of Velez is always positive in phlyctenular affections. (2) The results obtained from etiologic analysis of phlyctenular affections and from antituberculous treatment of the condition indicate that the disturbance is of tuberculous origin. (3) In all cases there was a perfect harmony among the different tests to which the patients were submitted (index of Velez, skin tests, radiographic investigation, and clinical examination). (4) All cases of phlyctenular keratoconjunctivitis must be treated as pretuberculous or tuberculous, unless the general condition of the patient indicates other treatment. (5) The value of the index of Velez ought to be more widely known among the medical profession.

Ramon Castroviejo.

Barrett, James. **Note on phlyctenular ophthalmia.** Brit. Jour. Ophth., 1939, v. 23, Oct., pp. 669-671.

From his experience the author questions whether the fact that phlyctenular and other ophthalmic cases give a tuberculin reaction is a certain indication that the cause is tuberculous, and asks whether both tubercle and phlyctenular ophthalmia alike may not be products of malnutrition. The affection was frequently noted in girls whose diet was largely bread, butter and tea, whereas with a full diet and local treatment the condition disappeared.

D. F. Harbridge.

Brasil do Amaral, N. M. **New orientations in the treatment of trachoma.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 2, pp. 671-673.

The author has found electrocoagulation of the granulations an excellent procedure in the treatment of trachomatous conjunctivitis.

Ramon Castroviejo.

Busacca, A. **Research about trachoma.** Trabalhos do Primeiro Cong.

Brasileiro de Ophth., 1936, v. 2, pp. 738-739.

This is a brief comment on the etiology, pathogenesis, and treatment of trachoma. The author believes that trachomatous pannus is produced by localization of the agent of trachoma in the cornea itself. A method of treating trachoma is mentioned, by which the author claims to have obtained cures in every case treated.

Ramon Castroviejo.

Cordero, Celso. **Coefficients of correlation between the morbidity of the conjunctiva and some meteorologic conditions.** Arch. di Ottal., 1939, v. 46, May-June, p. 133.

A method of higher mathematics used in statistical compilations has been applied to the study of the morbidity of the conjunctiva as related to meteorologic conditions. The author cites many examples of meteorologic factors producing disease. In this study a period of 27 years is considered and the following groups are analyzed: (1) conjunctivitis of Koch-Weeks, (2) conjunctivitis of diplobacillus of Morax-Axenfeld, (3) conjunctivitis of pneumococcus, and (4) other forms of conjunctivitis. The author concludes that there is a rise and fall of the infections during certain months of the year and under certain climatic conditions.

H. D. Scarney.

Feigenbaum, Aryeh. **Notes on unilateral trachoma and on the question of immunity in trachoma.** Acta Ophth. Orientalia, 1938, v. 1, Dec., p. 103.

Systematic examination carried on by the author proved that in unequally severe trachomatous changes in the two eyes chronic nasal disorders were present in 68 percent of the cases on the side corresponding to the more severely affected eye. The author assumes that reflex atony of the conjunctival vessels, originating from a chronic catarrhal irritation of the nasal cavity, impaired the nutrition of the conjunctiva, which thus became less resistant to infection. Unilateral trachoma may also be produced by a weakened virus. In these cases the natural resistance of the tissues in the healthy side of the nose prevented the weakened virus from acting upon the eye.

R. Grunfeld.

Griffey, E. W. **Rhinosporidiosis.** Amer. Jour. Ophth., 1939, v. 22, Dec., pp. 1389-1390.

Hatchek, G. **The efficacy of various methods in the treatment of trachoma.** Acta Ophth. Orientalia, 1939, v. 1, April, p. 152.

The various commonly used trachoma treatments are reviewed. In florid follicular trachoma the follicles are gently scarified or scraped with a sharp knife. The acute irritative state of the trachomatous pannus is treated as a corneal affection. The lids are painted with 5-percent argyrol; prontosil rubrum, milk injections, and vitamin A are administered. The lacrimal canal is frequently syringed but all energetic measures such as massage, peritomy, and scarification are to be postponed. For the prevention of recurrence in chronic pannus, excision of the tarsus and peritomy are efficacious. The chronic inflammatory pannus heals quickly after energetic treatment of the conjunctiva. In cases of inveterate connective-tissue degeneration of the pannus, alcohol treatment (Streiff) and ginger treatment give good results.

R. Grunfeld.

Hatchek, G. **Experiences with different sulphanilamide preparations in**

the treatment of trachoma. Acta Ophth. Orientalia, 1939, v. 1, July, p. 243.

The author asserts that prontosil rubrum is the most effective sulphanilamide preparation in the treatment of trachoma and is most efficacious when used both orally and parenterally.

R. Grunfeld.

Julianelle, L. A. **The antigenicity of the virus of trachoma.** Amer. Jour. Path., 1939, v. 15, May, p. 279.

After describing methods of experimentation, the author comes to these conclusions: (1) Clinical observation reveals very little if any immunity to trachoma. (2) It has not been possible to demonstrate increased resistance to experimental trachoma in monkeys following recovery from the infection. (3) The serum or defibrinated blood of patients with active infections of varying duration exerts no neutralizing or protective effect on the virus of trachoma. (4) The serum of infected or recovered monkeys contains no antibodies demonstrable by the usual methods of detection. (5) Likewise, the serum of rabbits or susceptible monkeys receiving repeated intravenous injections of active trachomatous tissues contains no antiviral substances. (6) It is concluded that the virus of trachoma is an impotent and ineffectual antigen. (3 references.) Ralph W. Danielson.

Julianelle, L. A. **A note on the immunology of trachoma.** Amer. Jour. Ophth., 1939, v. 22, Dec., pp. 1326-1328.

Julianelle, L. A., Lane, J. F., and Whitted, W. P. **The effect of sulphanilamide on the course of trachoma.** Amer. Jour. Ophth., 1939, v. 22, Nov., pp. 1244-1252.

Julianelle, L. A., Lane, J. F., and Whitted, W. P. **The effect of tartar emetic on the course of trachoma.** Amer. Jour. Ophth., 1939, v. 22, Dec., pp. 1390-1396.

Kondoyaroff, C. G. **Unilateral trachoma by slitlamp.** Acta Ophth. Orientalia, 1938, v. 1, Dec., p. 98.

Slitlamp examination is imperative for early diagnosis of fresh cases of trachoma and for retrospective recognition of cured cases. In 38 cases of so-called unilateral trachoma, the author observed fine cicatricial changes in the conjunctiva and obliterated vessels at the limbus of the healthy eye, indicating a latent trachomatous process. Neither unilateral trachoma nor unilateral immunity exists according to the author. The unequal course of the trachomatous process in the two eyes is maintained by a unilateral disease of the lacrimal apparatus, nose, or accessory sinuses. R. Grunfeld.

McGovern, F. H. **Parinaud's oculoglandular syndrome.** Amer. Jour. Ophth., 1939, v. 22, Dec., pp. 1400-1402.

Meyerhof, Max. **Remarks on trachoma healing without visible scar formation.** Acta Ophth. Orientalia, 1938, v. 1, Dec., p. 92.

The author observes that trachoma I and II (MacCallan) may heal without cicatrization. The previous presence of trachoma can be diagnosed from the presence of pannus with or without Herbert's pits. Scarless healing can be obtained even in certain cases of trachoma III by a daily superficial scarification of the follicles and by gentle rubbing of the conjunctiva with a 1 to 1000 sublimate, protargol, or hypertonic solution. Trachoma does not produce immunity, since the author repeatedly observed reinfection of completely cicatrized conjunctivas (trachoma IV). R. Grunfeld.

Olive Leite, Antonio. **Contribution to the treatment of trachoma.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 2, pp. 675-697.

In the treatment of trachoma the author uses a Brazilian preparation, "Karpotran," obtained by combining copper with fatty acids extracted from the "carpotroche brasiliense" (also called "chaulmoogra brasileira"). From this combination of oils and salts of copper is obtained a colloid of copper which is used in subconjunctival injections. In 22 cases very successful results were obtained after about 45 to 60 injections of the colloidal preparation. In some stubborn cases, the number of injections may be increased until cure results. Ramon Castroviejo.

Poleff, L. **Culture in vitro of the corpuscles of trachoma.** Brit. Jour. Ophth., 1939, v. 23, Nov., pp. 738-740.

In view of the uncertainty which exists as to the culture of trachoma corpuscles, the author presents a series of photomicrographs which exhibit typical pictures of the various stages of their evolution in vitro. Trachomatologists are concerned with a single minute infecting agent which has characteristics of both a virus and of a rickettsia. Poleff concludes that the rickettsia-like corpuscles described by Busacca and later by Cuénod are identical with the inclusions of Halberstaedter and Prowazek at certain evolutionary stages. (Photomicrographs.) D. F. Harbridge.

Poursines, Y., Froge, P., and Chiniara, J. **Contribution to the study of the pathologic anatomy and biomicroscopy of trachoma.** Acta Ophth. Orientalia, 1939, v. 1, April, p. 129, and July, p. 202.

The authors compared the biomicroscopic and histologic pictures in trachomatous conjunctivitis. (25 figures.) R. Grunfeld.

Shimkin, N. I. **Treatment of trachoma with sulphanilamide.** Acta Ophth. Orientalia, 1939, v. 1, July, p. 250.

The author treated seven cases of severe trachoma and achieved remarkable results. Treatment consisted of 15 grains of sulphanilamide three times daily for 10 to 14 days. A 5-percent prontosil-rubrum ointment was used simultaneously. No toxic effect was noted. R. Grunfeld.

Sie-Boen-Lian. **The use of Uliron in the treatment of gonorrheal conjunctivitis.** Ophthalmologica, 1939, v. 97, Aug., p. 341.

Uliron (4-[4′-aminobenzol-sulfanannido] benzol-sulfondimethylamid) has been widely and successfully used in urethritis. Its use in ophthalmic gonorrhea has been reported. The drug alone is very effective, but the best therapy has been found to be Uliron (two tablets on each of three successive days) combined with fever induced by intravenous typhoid vaccine, and irrigation with 1 to 4000 potassium permanganate solution. In fifty patients the conjunctiva became free from organisms in four days. F. Herbert Haessler.

Sniderman, H. R. **Larval conjunctivitis. (Oestrus ovis).** Amer. Jour. Ophth., 1939, v. 22, Nov., pp. 1253-1255.

Spearman, M. P., and Vandevere, W. E. **Sulfapyridine in trachoma.** Jour. Amer. Med. Assoc., 1939, v. 113, Nov. 11, p. 1807.

Two cases of trachoma which had proved intractable to all other methods of treatment, including sulphanilamide, showed remarkable remissions with

great improvement in vision following treatment with sulfapyridine.

George H. Stine.

Stern, H. J. **Spring catarrh and dryness of the air.** Acta Ophth. Orientalia, 1938, v. 1, Dec., p. 107.

The increased frequency of spring-catarrh cases during sirocco periods indicates that extreme dryness of the air and increased sunlight may be contributory agents in addition to an allergic disposition. R. Grunfeld.

Swan, K. C., and Allen, J. H. **Streptococcic pseudomembranous conjunctivitis treated with sulphanilamide.** Amer. Jour. Ophth., 1939, v. 22, Nov., pp. 1255-1261.

Trapezontzeva, E. I. **Trachoma and bacterium granulosis Noguchi.** Viestnik Opht., 1939, v. 15, pt. 1, p. 27.

This, the fifth article on the subject, deals with demonstration of bacillus Noguchi in filtrates of trachomatous material. Bacillus Noguchi can be isolated from filtrates of trachomatous material by filtering the suspensions through a Chamberlain no. 3 filter. The bacillus does not pass through a Chamberlain no. 5 filter. The number of bacterial elements passing through the filter is insignificant and filtrates of trachomatous material may be considered practically sterile. No other microbe was isolated from the trachomatous filtrates. Cultures of filtrates should be observed for several weeks or months. Ray K. Daily.

Trovati, Emma. **The vasomotor reaction of the conjunctiva under various luminous irradiations with photodynamic substances instilled in the fornix.** Ann. di Ottal., 1939, v. 67, Aug., p. 561.

The term "photodynamic" has been applied to substances which, when introduced into the organism and exposed to the action of light, produce special reactions. These substances are for the most part colored; some of them are of organic origin (such as bile pigment), and the effects produced depend on the substance itself and on the spectral band from which the radiation emanates as well as on the length of time of the exposure. The effects produced vary from simple congestion to intense edema or even necrosis of the tissue. Experiments with various substances were made both on man and on lower animals. Fluorescein especially seemed to sensitize the tissues to certain forms of irradiation. The hypothesis of the author is that certain substances in the organism determine and accentuate the phenomena produced on the bulbar conjunctiva under the action of the irradiation emanation from different spectral bands. (Bibliography.) Park Lewis.

6

CORNEA AND SCLERA

Argüello, D. M., and Tosi, B. **Vogt's exfoliation of the lens capsule associated with thrombosis of the temporal vein and Stähli's line.** Arch. de Oft. de Buenos Aires, 1938, v. 13, Dec., p. 681. (See Section 9, Crystalline lens.)

Ballantyne, A. J. **Punctate and annular corneal dystrophy.** Ophthalmologica, 1939, v. 97, Aug., p. 375.

Ballantyne replies graciously to Kraupa's polemic remarks (see below) but is not convinced by Kraupa's reasoning about the etiology of the lesion. F. Herbert Haessler.

Blanchi, Guido. **Band-like opacity of the cornea.** Rassegna Ital. d'Ottal., 1939, v. 8, July-Aug., p. 479.

Blanchi reviews the literature of the

subject and describes three cases of the secondary type which were studied clinically and histologically. The changes in the tissues consist in alterations of the spaces in the epithelium, which may be either increased or decreased; alterations of Bowman's membrane, including calcareous granules, degeneration, and fragmentation; degenerative changes of the parenchyma (colloid, amyloid, hyaline, and calcareous); and newly formed connective tissue, above or below Bowman's membrane, forming a true band across the cornea. Etiologically one must consider constitutional and local causes, including trauma, disturbances of internal secretion, and trophic changes. (4 figures.) Eugene M. Blake.

Boeck, J. **Experiments in correcting corneal astigmatism with electrocoagulation.** Wiener klin. Woch., 1939, v. 52, Oct. 27, pp. 971-974.

After giving a short historical summary of the surgical methods used to influence corneal astigmatism the author describes his method of corneal coagulation with the high-frequency current. Punctures are made with a needle 0.5 mm. in length and using a current of 20 to 30 milliamperes. They are placed 1 to 1.5 mm. inside the limbus at the two ends of the meridian of lesser refraction. These punctate coagulations increase the corneal curvature of the meridian in which they are placed. After the first few coagulations the amount of correction can easily be controlled by the use of Javal's ophthalmometer. The author has found that considerable initial overcorrection is necessary to insure a permanent result. After two or three months the overcorrection disappears, and in two or three more months some of the astigmatism in the original axis usually returns, but with the error considerably less than before operation. The procedure is painless and is tolerated with strikingly little postoperative reaction. The resulting scars are very delicate.

Bertha Klien.

Busacca, Archimède. **The study of the structure and nature of palisades, considered up to now as a network of perilimbal lymphatics.** Arch. d'Opht. etc., 1939, v. 3, Aug., p. 693.

By studying serial sections through the limbal region of an eye removed from a melanodermic individual, the author obtained evidence that the so-called pericorneal lymphatics and perivascular lymph spaces are actually papillary ridges of the mucous membrane (lamina propria) leveled by a thickness of epithelium. They correspond to the anatomic formations known as the papillary ridges of Manz. Usually each ridge has its own blood vessels and lymphatics. When pigment is found it is situated in the epithelial cells, especially the basal ones, along the sides of the ridges. (Photomicrographs, diagrams, bibliography.)

Derrick Vail.

Casini, Francesco. **Contribution to the question of the etiopathogenesis of keratoconus.** Arch. di Ottal., 1939, v. 46, May-June, p. 161.

The etiopathogenesis of keratoconus is considered on the basis of avitaminosis, constitutional hereditary abnormality, or possible relation to a glandular abnormality correlated with the hypophyso-diencephalic syndrome. The author carried out treatment with large doses of vitamins A and D and also employed calcium therapy. He believes that vitamins A and D have definite importance, but their lack is manifested

only where there is an inherited corneal weakness. H. D. Scarney.

Fuchs, A. **Primary zonular opacity of the cornea.** Klin. M. f. Augenh., 1939, v. 103, Sept., p. 300.

Without other local or general pathologic symptoms, a boy of 11 years had had for three years a zonular opacity of both corneae with intense formation of calcareous lumps. His father had been operated on for secondary glaucoma and there were considerable deposits of lime in the corneal scars and drusen in the discs. A sister showed extraordinary changes in corneal scars. The fact that three members of the same family had severe ocular affections indicates a general organic disposition or weakness.

C. Zimmermann.

Gelarie, A. J. **Experimental syphilitic keratitis in the rabbit.** Jour. Infectious Dis., 1939, v. 65, July-Aug., pp. 84-85.

In experimental syphilis of the rabbit, either a primary or a metastatic keratitis can be produced. The latter corresponds to the usual form of keratitis in man, and may be considered as a late or tertiary lesion developing as a response of the allergic tissue to the presence of small numbers of spirochetes. By serial passage from cornea to cornea, a considerable adaptation of the spirochetes to the corneal tissue was obtained by Gelarie; the positives increasing from 10 to 80 percent in eight transfers. T. E. Sanders.

Gorchakova, H. V., and Gorchakova, I. A. **Two cases of parenchymatous keratitis caused by trauma in congenital syphilitics.** Viestnik Opht., 1939, v. 15, pt. 1, p. 94.

In one case the attack followed a blow on the eye by an elbow, and in the other it followed an injury from a clod of earth. R. K. Daily.

Günther, Rosemarie, **Pathologic findings in a cornea affected by epidemic keratitis.** Klin. M. f. Augenh., 1939, v. 103, Sept., p. 309.

Histologic examination of the cornea of a man of 67 years with bilateral epidemic keratitis showed spotty lumps of decay on Bowman's membrane. Apparently infiltrations with lymphocytes, histiocytes, and fibroblasts had occurred under the epithelium, crowding it from Bowman's membrane and attenuating it. C. Zimmermann.

Hofe, K. vom. **Relations between macrocornea and buphthalmos.** Deut. Ophth. Gesel., 1938; in Klin. M. f. Augenh., 1938, v. 101, July, p. 105.

Clinical observations by the author show that juvenile megalocornea may develop into buphthalmos in later years. Both forms may be present in the same individual, the two eyes being affected differently. Both conditions may under certain circumstances occur alternately in children of the same family. The two developmental anomalies are not basically different but show, on the contrary, close genetic relations to each other.

Percy Fridenberg.

Knapp, H. C. **Dystrophia adiposa corneae.** Amer. Jour. Ophth., 1939, v. 22, Nov., pp. 1239-1243.

Kraupa, Ernst. **Punctate and annular corneal dystrophy.** Ophthalmologica, 1939, v. 97, Aug., p. 374.

Kraupa replies to Ballantyne's criticism (Amer. Jour. Ophth., 1939, v. 22, 1940). He points out that the punctate lesion described by Ballantyne resembles that of Kraupa's first case, and the annular lesion described by Coats re-

sembles that of Kraupa's second case. Considering these experiences in retrospect, Kraupa believes that the dystrophy is a secondary reaction to foreign-body injury. He bases this conclusion on the fact that the lesions may occur at any age, are usually of the same size, appear in two different but associated forms, remain unaltered for years, and are not related to changes in the corneal epithelium, endothelium, or nerves.

F. Herbert Haessler.

Loehlein, M. **Progress in the field of corneal transplantation.** Deut. Ophth. Gesell., 1938; in Klin. M. f. Augenh., 1938, v. 101, July, p. 106.

In all but one of 18 cases, the transplants were successful. The single exception, a case of lime burn with secondary glaucoma and faulty light projection, went on to phthisis bulbi. In no case was the final vision less than that noted before transplantation. In two cases of repeated relapses of corneal fistula following keratitis, firm union was finally obtained after punching out the diseased area. Transplantation as a prophylactic measure may be beneficial where the cornea is threatened by severe processes such as malignant ulcus serpens, rosacea keratitis, or even pyocyaneus ulcer involving the entire cornea. Loehlein uses a 3 to 5 mm. trephine and Schoeler's inverted conjunctival flap. Cadaveric transplants previously fixed in formalin are quite as good as the living tissue and are even better than the simple cadaveric material. Percy Fridenberg.

Orlov, K. X. **Dystrophy of the cornea.** Viestnik Opht., 1939, v. 15, pt. 1, p. 3.

A report of a case. The corneal disturbance consisted in the formation of hyperplastic epithelial nodules composed of 35 layers of corneal epithelium, and in the transformation of some of the superficial epithelium into a cutaneous-like tissue. Some of these nodules were easily removed from the cornea, and proved microscopically to be of multilayered epithelium. None of the therapeutic or surgical procedures tried gave more than temporary relief. (Illustrations.) Ray K. Daily.

Orlov, K. X. **Keratokeras.** Viestnik Opht., 1939, v. 15, pt. 1, p. 6.

A report of a case of horny formation on the surface of a staphylomatous cornea. The formation was loosely bound to the cornea, and crumbled while the eyeball was being exenterated. The microscopic picture demonstrated gradual transformation of epithelial cells into homogeneous tissue, the accumulation of which under the influence of dust and desiccation formed a firm corneal neoplasm. (Photomicrographs.) Ray K. Daily.

Pincus, M. H. **Krukenberg spindles in a patient with interstitial keratitis.** Amer. Jour. Ophth., 1939, v. 22, Dec., pp. 1397-1399.

Schmidt, R. **Comparative investigations on the action of short waves and of electric pads in different processes of the corneae of rabbits.** Klin. M. f. Augenh., 1939, v. 103, Sept., p. 314.

No essential difference in the effectiveness of the two methods could be ascertained in corneal ulcers produced by staphylococcus albus. Corneal processes induced by herpes virus and pneumococci remained uninfluenced by treatment with either short waves or electric pads, undoubtedly because of the high virulence of the organisms.

C. Zimmermann.

Schupfer, Francesco. **Research on the influence of testicular extract on the permeability of the cornea. Experience with ascorbic acid.** Ann. di Ottal., 1939, v. 67, July, p. 527.

It has been shown by various research workers that testicular extract contains in large quantity a substance that increases the permeability of the dermal tissues. After the instillation of testicular extract, the author studied the passage of vitamin C from the conjunctival sac into the aqueous. He succeeded in recovering in the aqueous a very definite amount of ascorbic acid while under normal conditions the amount present is minimal. This increase of permeability continued for 24 hours, though in reduced amount, after the instillation of the testicular extract. The author further discovered that if the extract was dialyzed through cellophane, the permeability of the tissue was increased still more while at the same time the action of the extract was rendered less irritative. (Bibliography.)

Park Lewis.

Shimkin, N. I. **Resection of anterior staphyloma of the cornea by means of gradual incisions.** Brit. Jour. Ophth., 1939, v. 23, Oct., p. 671.

In Palestine the author observed many patients with anterior staphyloma of the cornea. Due to the fact that patients demanded retention of the globe, resection of the cornea was practiced. A complete resection was not done at once, but by gradual incisions of about 2 mm. each. A horizontal oval area of cornea to be resected was outlined and with the sharp point of a scalpel the end of the ovoid was cut for a distance of about 1.5 mm. Then a suture was placed and tied, and this was repeated every 2 mm. until the entire resected cornea was removed. In all, about seven sutures were required. By this method loss of vitreous is minimized and the lens is not disturbed. The author has operated upon 31 patients with satisfactory results.

D. F. Harbridge.

Tai, A. L. **A new instrument for keratoplasty.** Amer. Jour. Ophth., 1939, v. 22, Dec., pp. 1402-1403.

Trovati, Emma. **Experimental and clinical research on the action of ultraviolet ray on the normal eye and in certain corneal affections.** Ann. di Ottal., 1939, v. 67, July, p. 481.

Experiments were made on the normal eye with mercury-vapor and carbon-filament lamps. The intensity of the reaction was found to be directly proportionate to the length of the exposure. A short irradiation of five to ten minutes caused no immediate reaction. An exposure of half an hour to the rays caused irritative phenomena (photophobia, lacrimation, ocular congestion) to appear six hours later. These gradually disappeared, leaving no aftereffects. Marked improvement from irradiation was shown in various forms of vascular keratitis and in trachomatous pannus. The ocular tension showed itself in primary increase and secondary lowering. (6 figures, bibliography.) Park Lewis.

Vakker, A. G. **The use of chloricide in serpiginous ulcers and other ocular diseases.** Viestnik Opht., 1939, v. 15, pt. 1, p. 68.

A report of clinical and laboratory study. Chloricide is a solution containing 120 to 200 mg. of free chlorine per liter. Applications of the solution to cultures of staphylococcus aureus and albus and streptococcus hemolyticus produced destruction of the organisms within five minutes. Clinically, chlori-

cide was tried with satisfactory results in 36 ocular infections, eight of which were serpiginous ulcers. The solution is cheap and easily prepared.

Ray K. Daily.

Van Canneyt, J. **Comparative receptivity of different regions of the cornea of the rabbit to infection with syphilis.** Bull. Soc. Belge d'Opht., 1939, no. 78, p. 152.

The authors state as a result of their experiments that in the rabbit the superior segment of the cornea is the most sensitive to syphilitic infection, the inferior segment to a much less degree. The other zones become invaded by general infection only. They believe these facts depend upon the anatomic structure of the cornea, possibly upon the arrangement of the lymph spaces. Jerome B. Thomas.

Von der Heydt, R. **Corneal dystrophy and keratoplasty.** Schweizer med. Woch., 1939, no. 43, Oct., p. 1063.

Von der Heydt reports a case which he had had under observation for 15 years. The dystrophy consisted of a central disc-shaped area with a ground-glass appearance similar to the Bueckler type. Although the dystrophy was dominant, four brothers and one sister showed normal corneae. During the 15 years the vision decreased from 20/80 to 4/200. Castroviejo did a 57-mm. penetrating keratoplasty which had remained clear at the last observation, six months after the operation. The cornea was taken from the eye of a stillborn infant. Corrected vision was 20/80 and with a contact glass 20/60, but this latter was impractical for the patient. The author recommends keratoplasty if done by a capable surgeon with experience in that field.

Theodore M. Shapira.

Zolotareva, M. M. **Treatment of serpiginous ulcer by trepanation after Zonderman.** Viestnik Opht., 1939, v. 15, pt. 1, p. 64.

Zolotareva reports nine cases of severe serpiginous ulcer treated, with good results, by trephining through the center of the ulcer. This procedure is indicated in central ulcers accompanied by hypertension; it promotes the escape of pus and maintains hypotension long enough to aid recovery.

Ray K. Daily.

7

UVEAL TRACT, SYMPATHETIC DISEASE, AND AQUEOUS HUMOR

Alaerts, L. **The rhythm of dilatation of the pupil induced by medicaments.** Bull. Soc. Belge d'Opht., 1939, no. 78, p. 91.

By the term rhythm the author wishes to express in one word the speed, duration, and amplitude of the movement. His study relates to the slow reaction of the iris to medicaments, or a static condition of the pupil at different stages of its dilatation. The subjects chosen for experiment had normal health and vision and were examined under the same light conditions. The pupils were measured before instillation of the collyrium and then every few minutes up to maximum dilatation. The figures were reduced to graphic curves, which serve to illustrate the report. The drugs used were cocaine, adrenalin, ephedrine, and homatropine, administered alone or in various combinations. (8 references.)

Jerome B. Thomas.

Bistis, J. **Atony of the pupil and Adie's syndrome.** Arch. d'Opht. etc., 1939, v. 3, Aug., p. 706.

Pupillary atony is characterized by

mydriasis, abolition of light reflex, slow contraction of the pupil to convergence, and still slower dilatation. A case is reported of this condition, and Adie's syndrome is discussed. The author believes that the tonic pupil in his case was due to an irritation of the sympathetic nerves as a result of a chronic pharyngeal inflammation following tonsillectomy. Derrick Vail.

Fehrmann, Horst. **Diabetic rubeosis of the iris and its constitutional meaning.** Graefe's Arch., 1939, v. 140, pt. 2, pp. 354-371.

Search of the literature revealed reports of eleven cases, with histologic study in one instance. The author made microscopic studies of both eyes of one patient, as well as clinical observations of two other cases. He also examined two patients who had glaucoma and new-formed vessels on the iris, studying one eye histologically. His findings and those reported in the literature lead him to consider diabetic rubeosis of the iris as a special disease of the eye in diabetes mellitus. The involved eyes showed a network of small capillary vessels and loops on the anterior surface of the iris. These vessels were most numerous in the region of the sphincter and in the periphery, where they appeared to arise from the angle of the anterior chamber. Acute uncompensated glaucoma had developed. Miotics made the condition worse, and even precipitated an acute attack of glaucoma. New-formed vessels were found upon the optic nerve-head and on the retina beneath the internal limiting membrane. Pathologic sections showed relatively wide, new-formed capillaries unaccompanied by connective tissue, in and upon the anterior border layer of the iris. These vessels were found in the peripheral synechia, a finding not seen in cases of primary glaucoma with vessels on the iris. There is no effective therapy, and no operative procedure produces a lasting reduction in tension. Differential diagnosis between rubeosis and new-formed vessels on the iris in glaucoma patients without diabetes can be made clinically and anatomically. The latter vessels are much larger and thicker, run in a connective tissue laid down on the anterior surface of the iris, and sometimes pass the pupillary border.

The almost regular occurrence of diabetic retinopathy in these cases suggests a similar etiology, based upon increased permeability of vessels and a disturbed tissue metabolism. The course is downhill with painful glaucoma and blindness following the primary changes in the iris. Since the condition appears in both young and old patients, and with either mild or severe diabetes, it bears no relation to prognosis as to life. Charles A. Perera.

Krueckmann. **Influence on the eye of pulmonary reinfection and superinfection.** Deut. Ophth. Gesell., 1938; in Klin. M. f. Augenh., 1938, v. 101, July, p. 101.

A preliminary report definitely showing reinfection of a lung with secondary iritis. Percy Fridenberg.

Matzdorff, P. **Contributions to the knowledge of Adie's syndrome.** Deut. med. Woch., 1939, no. 33, Aug., p. 1307.

The author cites five clinical cases of Adie's syndrome (tonic pupils and absent tendon reflexes). Although the syndrome has never been reported in central-nervous-system lues, Matzdorff describes one case in which lues was found. Another case followed diphtheria.

Theodore M. Shapira.

Simoes, Elysio. **Tuberculosis of the uveal tract.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 2, pp. 621-625.

This is a report of two cases of tuberculosis of the uveal tract. The first case was of iritis and was treated with salts of gold and injections of calcium. The second case was of uveitis and was treated with tuberculin. In each case, after considerable improvement, the patient failed to return for further treatment. Ramon Castroviejo.

Zimin, V. I. **Therapy of the reflex vegetative system in the treatment of iritis and iridocyclitis.** Viestnik Opht., 1939, v. 15, pt. 1, p. 79.

Mud baths and galvanization of the cervical vegetative apparatus were used with very good results in 19 cases of iritis and nine cases of iridocyclitis. Atropine was the only other therapeutic agent used in the nonluetic cases; the luetic cases were given specific therapy. The conclusions are that this form of therapy shortens the period of disability and can be administered as an ambulatory procedure.

Ray K. Daily.

8

GLAUCOMA AND OCULAR TENSION

Cavka, V. **The study of hyperglycemia and hypoglycemia in relation to intraocular tension.** Ann. d'Ocul., 1939, v. 176, Aug., pp. 616-623.

The well-known ocular hypotony in cases of diabetic coma is reviewed. Very little is known about intraocular tension in hypoglycemia. Twenty-two cases of schizophrenia treated by insulin shock were examined. The ocular findings consisted of (1) blepharospasm apparently related to the myoclonic convulsions and therefore assumed to be the result of central irritation; (2) alternating miosis and mydriasis at intervals of from 30 seconds to 3 minutes, also assumed to be the result of central irritation; (3) dilatation of the retinal veins without change in the optic nerve-head; and (4) a definite increase in intraocular tension, never a decrease. The last finding was assumed to be the result of the alteration in water balance produced by large doses of insulin. John M. McLean.

Hofe, K. vom. **Relations between macrocornea and buphthalmos.** Deut. Ophth. Gesell., 1938; in Klin. M. f. Augenh., 1938, v. 101, July, p. 105. (See Section 6, Cornea and sclera.)

Rintelin, F. **Concerning the meaning of the word "glaucoma."** Schweizer med. Woch., 1939, no. 28, July, p. 646.

An etymologic study of the word "glaucoma" and its misuse in textbooks.. Theodore M. Shapira.

Rossel, S. I. **The effect of various concentrations of pilocarpine on intraocular tension.** Viestnik Opht., 1939, v. 15, pt. 1, p. 48.

Study of 44 glaucoma patients and five normal persons indicated that strong solutions of pilocarpine exerted no effect on normal eyes, that 0.5 to 3 percent solutions affected the glaucomatous eye in equal degree, and that 6 percent solutions had a more profound effect on intraocular tension after weaker solutions had failed.

Ray K. Daily.

9

CRYSTALLINE LENS

Knuesel, O. **Three cases of cataract due to electric current.** Schweizer med. Woch., 1939, Nov. 4., p. 1084.

The author reports three cases of electric cataract, two following trauma

from 50,000 volts and one after lightning. In each case characteristic lens changes were present. Under the anterior capsule were opacities which peripherally were pointed and axially appeared as a spider web or net. Under the posterior capsule was a corresponding opaque zone. Missing were the large subcapsular vacuoles and deposits of shiny appearance. The effect of the current is to inhibit growth of the young lens fibers, which degenerate by absorption of water. One case reported occurred 18 years after the original trauma. Theodore M. Shapira.

Varshavskii, I. K. **Intracapsular cataract extraction.** Viestnik Opht., 1939, v. 15, pt. 1, p. 8.

A defense of this surgical procedure and a plea for the manufacture of capsule forceps, which are at present practically unavailable in the Soviet Union. Ray K. Daily.

10

RETINA AND VITREOUS

Basile, Giambattista. **The biomicroscopy of subjects affected with retinitis pigmentosa.** Ann. di Ottal., 1939, v. 67, Aug., p. 587.

The author examined biomicroscopically the crystalline lenses of 26 patients suffering from retinitis pigmentosa. In those having incipient retinal degeneration there was slight clouding of the posterior subcapsular region, with punctiform opacities in the anterior cortical striae and brilliant iridescence of the posterior portion of the lens in some cases. When the degeneration was more advanced, the posterior polar opacity was more dense and took on a grayish-yellow color like that of fresh bread. The opacity became smoky toward the periphery of the lens but more frequently extended in radial opacities to the posterior pole. With this were observed grayish punctiform opacities frequently extending through the cortex to the anterior pole. The opacities, especially near the posterior pole, assumed a porous appearance and were of a yellowish tint. Toward the periphery these opacities took the form of dotted rays. In the more advanced form of degeneration the opacities were more abundant around the nucleus and the posterior suture. In only 12 of the 26 subjects were stellate opacities found in the posterior subcapsular area. In most of the cases the striae involved the anterior and posterior cortex extending to the nucleus and to the posterior suture. (One plate, bibliography.)

Park Lewis.

Borsotti, I., and Pillet, P. **Examination of the fundus during blood transfusions.** Ann. d'Ocul., 1939, v. 176, Aug., pp. 586-604.

The authors studied the retinal circulation in relation to systemic blood pressure during blood transfusions. The only outstanding findings were related to the central retinal vein. The cases fell into two general groups. One comprised those in which the systemic blood pressure showed a tendency to rise. In these patients there was either no change in the retinal veins or a disappearance of the spontaneous venous pulsation. The other group was of those patients in which the blood pressure tended to fall. In these there was either appearance of a retinal venous pulsation or augmentation of it if already present. John M. McLean.

NEWS ITEMS

Edited by Dr. H. Rommel Hildreth
640 S. Kingshighway, Saint Louis

News items should reach the Editor by the twelfth of the month.

Deaths

Dr. Patrick Chalmers Jameson, Brooklyn, New York, died October 27, 1939, aged 72 years.

Dr. Alfred Bielschowsky, died at Brooklyn, New York, on January 6, 1940, aged 68 years.

Dr. John Algernon Cavanaugh, Chicago, died November 17, 1939, aged 60 years.

Miscellaneous

The George Washington University School of Medicine, Washington, D.C., announces a Postgraduate Course in Aviation Ophthalmology and Aviation Medicine for graduates in medicine, April 1-6, 1940, inclusive. This course on aviation ophthalmology and aviation medicine is part of the postgraduate course in ophthalmology given from March 19-30, 1940, inclusive.

The Gill Memorial Eye, Ear, and Throat Hospital, Roanoke, Virginia, presents its Fourteenth Annual Spring Graduate Course in Ophthalmology and Otolaryngology from April 1 to 6, 1940. The following doctors will appear on the program: Dr. Karl S. Blackwell, Richmond, Virginia; Dr. Grady Clay, Atlanta, Georgia; Dr. C. C. Coleman, Richmond, Virginia; Dr. Parker Heath, Detroit, Michigan; Dr. H. S. Hedges, Charlottesville, Virginia; Dr. Wendell L. Hughes, Hempstead, Long Island, New York; Dr. Chevalier L. Jackson, Philadelphia, Pennsylvania; Dr. Peter C. Kronfeld, Chicago, Illinois; Dr. Dean M. Lierle, Iowa City, Iowa; Dr. Lyman Richards, Boston, Massachusetts; Dr. William F. Rienhoff, Jr., Dr. Harry R. Slack, Jr., Dr. Frank B. Walsh, Baltimore, Maryland; Dr. C. R. Straatsma, Dr. D. Blair Sulouff, New York, New York.

The Annual Graduate Course in Neuromuscular anomalies of the eyes, by Dr. George P. Guibor, Children's Memorial Hospital, Chicago, will be given February 18 to 23, 1940, inclusive.

The Mackenzie Post-Graduate Medical School conducted at St. Luke's Children's Hospital, Philadelphia, announces an intensive postgraduate course in several subjects including refraction, ophthalmic surgery, clinical ophthalmology, and ophthalmo-oto-neurology. These courses begin February 19, 1940, under the direction of George W. Mackenzie, M.D., 269 South 19th Street, Philadelphia.

St. Luke's Hospital, New York, announces an 18-months' residency in ophthalmology (and discontinuance of its combined residency in ophthalmology and otolaryngology, formerly offered). The service will commence on January 1, annually. For the first six months the appointee will be enabled through the hospital's affiliation to take the full-time course of instruction in basic sciences and principles of ophthalmology given at the Graduate School of the College of Physicians and Surgeons of Columbia University. During the entire residency of 18 months, accommodation will be furnished in the hospital. A small monetary allowance will be paid during the clinical portion of the service.

Upon the termination of the hospital service, the graduating resident will be assisted in securing a clinical appointment to complete the requirements for the American Board of Ophthalmology.

This residency offers an active ward, clinic, and private ophthalmological service, with the reciprocal consulting facilities in all departments of medicine which only a large general hospital can provide.

Societies

A Pan-American Congress of Ophthalmology will be held in Cleveland on October 11 and 12, 1940. In order to facilitate the institution of this congress, the American Academy of Ophthalmology and Oto-Laryngology has undertaken the necessary arrangements. The official languages of the congress will be Spanish, Portuguese, and English, and the committee members in charge of developing the program are Dr. Conrad Berens of New York City and Dr. M. Alvaro of São Paulo, Brazil. All ophthalmologists are eligible to membership in the congress upon registration and payment of the $5.00 fee. Until the congress committee is in session, all business pertaining to the congress can be transacted through Dr. W. P. Wherry, executive secretary of the American Academy of Ophthalmology and Oto-Laryngology.

Individual invitations to attend the congress will not be sent to ophthalmologists in the United States and Canada. This notice will be taken as the official invitation.

It was recently decided that the next meeting of the Pacific Coast Oto-Ophthalmological Society will be held in Spokane, June 24, 25, 26, and 27, 1940. Dr. Frederick G. Sprowl will be the president.